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WEIL'S DISEASE IN BRISBANE.

By D. W. JOHNSON, M.B., B.S., H. E. BROWN and E. H. DERRICK, M.D.,

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IN 1886 Weil described four cases of infectious jaundice. Since then the symptom complex of fever, jaundice, enlargement of liver and spleen, hæmorrhage and occasional febrile relapse has been commonly called Weil's disease. In 1914 Inada and Ido discovered a spirochæte-like organism in a case of Weil's disease in Japan, and named it *Spirochæta icterohæmorrhagiæ*. Similar organisms were found independently soon afterwards in Germany, and many cases of jaundice in Europe have since been found to have a spirochætal origin. Most subsequent writers have accepted the identity of the Japanese and European organisms. Weil's disease, like its chief carrier, the sewer rat, has a world-wide distribution.

In 1917 Noguchi introduced the generic name *Leptospira*.

Following up this work investigators in many countries have found numerous cases of fever, with and without jaundice, to be due to organisms of the genus *Leptospira*. It has become evident that *Leptospira icterohæmorrhagiæ* is only one of a large group of leptospiræ pathogenic to man. Many of these leptospiræ have been given specific names, such as *Leptospira canicola*, *Leptospira autumnalis*, *Leptospira hebdomadis et cetera*; others are not yet definitely classified. They are all morphologically and culturally similar, and may be distinguished only by serological means.

The term Weil's disease, used in a broad sense, has been applied to any form of infectious jaundice caused by leptospiræ. In order to avoid confusion, the more recent practice is to restrict this term to the classical type caused by *Leptospira ictero-*

hæmorrhagiæ, and to use the inclusive term leptospirosis for the leptospiral infections generally.

Leptospirosis, as it occurs in north Queensland, is generally called Weil's disease, but the applicability of the term is questionable. The infecting leptospiræ are still in process of classification, and the exact part played by *Leptospira icterohæmorrhagiæ* in the north remains to be established.

The present article shows that Weil's disease, in the strict sense, is definitely present in Australia. Three cases have occurred in Brisbane, but one of the patients was infected whilst in north Queensland. In addition, *Leptospira icterohæmorrhagiæ* has been recovered from a number of Brisbane rats.

Case I.

Clinical History.

The following clinical history is taken from notes kindly supplied by Dr. P. J. Kelly.

E.O., a male, aged twenty-seven years, had been employed for seven months cleaning sewers. He had been working in one particular storm-water sewer for a fortnight, when, on August 30, 1935, he became ill, with pains in the abdomen and vomiting. A severe headache followed and pains all over. The temperature rose to 39·4° C. On the sixth day jaundice came on, and during the next few days the patient was quite ill. There was some epistaxis.

On September 9, the eleventh day of his illness, he was admitted to the Mater Misericordiæ Public Hospital for investigation. He was then deeply jaundiced. There was a hæmorrhagic bleb on a finger. There were scattered rhonchi throughout the lungs. The liver was not enlarged, nor the spleen palpable. By the time of his admission to hospital the temperature had returned to normal, where it remained, except for an evening rise on the twenty-seventh, twenty-eighth and twenty-ninth days of illness (to 37·5°, 37·4° and 37·9° C. respectively). The jaundice gradually lessened, the general strength improved, and he was discharged from hospital on October 2. He resumed work after an absence of eight weeks.

Pathological investigations as shown in Table I were carried out during the illness, some of them in the Health Department laboratory, some in the hospital laboratory.

Clinically the case was a typical one of Weil's disease, and this diagnosis was strongly supported by the man's occupation. It was disappointing, therefore, that all the investigations at the time gave negative results. Only one strain of leptospira, a north Queensland one, was then available in Brisbane for agglutination work. The patient's serum failed to agglutinate this.

Reinvestigation.

The acquisition recently from Dr. Mertens, of Batavia, of a wide range of leptospiral types, and especially the isolation of strains from Brisbane rats, called for the reinvestigation of the patient. Accordingly, a sample of blood was obtained from him on February 5, 1937, that is, seventeen months after the onset of his illness.

With this serum, agglutination tests according to the technique recommended by Davidson, Campbell, Rae and Smith,[1] were performed against all the available strains of leptospiræ. Subsequently a human strain (J.J.D.) was obtained locally and it also was tested. The cultures used were live cultures, four to six days old, grown in Schüffner's medium.[2] Readings were made after three and a half hours at 30° C. The results are shown in Table II.

Comment.

After an attack of Weil's disease residual agglutinins are known to persist in the serum for several years. Postmus[4] demonstrated their presence 6,066 days after the onset of the disease, and Fairley[5] showed agglutinins to be present twelve and a half years after recovery. This persistence of agglutinins is an important phenomenon

TABLE I.

E.O., Pathological Investigations, September, 1935.

Day of Illness.	Material Examined.	Test.	Result.
8	Blood, 3·5 mils.	Inoculation of guinea-pig.	No reaction.
11	Blood.	Culture.[1]	No growth.
	Blood, 1 mil.	Inoculation of guinea-pig.[1]	No reaction.
	Urine, 2 mils.	Inoculation of guinea-pig.[1]	No reaction.
12	Urine, centrifuged deposit.	Dark-ground examination.	No leptospiræ seen.
		Inoculation of guinea-pig.	No reaction.
16	Blood.	White cell count.[1]	5,900. Neutrophile cells, 63; lymphocytes, 36; eosinophile cell, 1.
		Red cell count.[1]	4,350,000.
		Hæmoglobin estimation.[1]	84%.
	Serum.	Van den Bergh test.[1]	Immediate direct reaction.
18	Urine.	Dark-ground examination.	No leptospiræ seen.
		Inoculation of guinea-pig.	No reaction.
	Serum.	Agglutination, leptospira.	No agglutination.
		Agglutination, *Eberthella typhosa*.	No agglutination.
		Agglutination, *Salmonella paratyphi*.	No agglutination.
		Agglutination, *Salmonella schottmülleri*.	No agglutination.
		Agglutination, *Proteus* X 19 and XK.	No agglutination.
		Agglutination, *Brucella abortus*.	No agglutination.

[1] These investigations were done in the laboratory of the Mater Misericordiæ Public Hospital. (Dr. G. Taylor.)

TABLE II.

Agglutination Tests with Serum of B.O., February, 1937.

Strain of Leptospira.	Dilution of Serum.							Control.
	1/10	1/100	1/200	1/800	1/1600	1/3200	1/12800	
Wijnberg. Classic Weil's disease type, Netherlands	+++	++	++	±	−	−	−	−
J.J.D. Human strain, Brisbane	+++	++	+	+	−	−	−	−
Rat 101. Rat strain, Brisbane	lysis	++	++	±	−	−	−	−
	+							
Rat 102. Rat strain, Brisbane	+++	++	++	+	±	−	−	−
Ballico. Human strain, north Queensland	++	+	−	−	−	−	−	−
Pomona. Human strain, Pomona, Queensland	−	−	−	−	−	−	−	−
Andaman. Human strain	+	±	−	−	−	−	−	−
Roesel. Dog type, Netherlands	+	±	−	−	−	−	−	−
Moscow. Russian slime fever type	+	−	−	−	−	−	−	−
Palembang. Rachmat type, Sumatra	±	−	−	−	−	−	−	−
Bangkinang. Rachmat type, Sumatra	+	−	−	−	−	−	−	−
Baermann. Sumatra strain	±	−	−	−	−	−	−	−

The Ballico strain was received from Dr. G. F. Lumley, Townsville; the exotic strains from Dr. W. K. Mertens, Batavia. For the origin of the Pomona strain, see reference (3).

and allows a retrospective diagnosis to be made even at a considerable time after the illness.

Agglutinins against leptospiræ are not found in the serum of healthy people taken at random, so that their presence is a reliable criterion of a previous infection. If a patient has had several illnesses in the past, a positive result to an agglutination test alone does not indicate which of them was Weil's disease; for this the test must be considered in conjunction with the clinical history.

In this case seventeen months had elapsed since the illness, yet the serum still agglutinated to a diagnostic titre leptospiræ of the classical Weil's disease type as well as the leptospiræ recovered from two Brisbane rats. This, taken in conjunction with the typical history, establishes definitely the nature of the illness of 1935 and vindicates the clinical judgement of Dr. Kelly, who felt sure at the time that his patient was suffering from Weil's disease.

Case II.

Clinical History.

The notes of the clinical history in this case are available by courtesy of Dr. A. R. K. Burne and Dr. L. J. Jarvis Nye.

R.K., a male, aged twenty-four years, was a plumber's assistant at the sugar mill, Babinda, north Queensland, from June until December 23, 1936. On that date he left Babinda and went to Oak Forest, near Kuranda, north Queensland. On January 5, 1937, he departed for Brisbane, arriving there on January 8.

On January 11, 1937, the patient developed a sore throat, with pyrexia (39·4° C.), headache and generalized muscular pains. The throat condition was described by Dr. Burne as a typical acute follicular tonsillitis. The throat condition improved in four or five days, but the patient's general condition deteriorated rapidly. On January 17 his friends noticed faint jaundice. The patient then had a collapsed appearance, with a dry tongue. The tonsils were mildly inflamed. The temperature was 36·1° C. The pulse rate was 112. The abdomen was slightly distended and the muscles were too rigid to palpate the spleen. There was no rash. No joint soreness was present, but the calves were tender on pressure. The knee jerks were sluggish and the abdominal reflexes absent. The urine contained a heavy cloud of albumin.

On January 18 the patient was seen in consultation by Dr. Nye and was transferred to a private hospital, where, although his temperature was never elevated, he became rapidly worse. Jaundice increased until it was intense, and the patient was unable to retain solid food by mouth. He became weak and listless, distension increased, and he died on January 23. No *post mortem* examination was performed. The clinical diagnosis was Weil's disease.

Pathological Investigations.

The following pathological investigations were carried out.

1. On January 18, 1937, serum was investigated and no agglutination was obtained with *Eberthella typhosa*, *Salmonella paratyphi* (paratyphoid A) and *Salmonella schottmülleri* (paratyphoid B).

2. On January 18, 1937, the Van den Bergh test gave an immediate direct reaction.

3. On January 18, 1937, a blood count was made. Red blood corpuscles numbered 3,480,000 and the leucocytes 17,600 per cubic millimetre (polymorphonuclear cells 97%, lymphocytes 3%). (Tests 1, 2 and 3 were performed by Dr. N. Gutteridge.)

4. On January 22, 1937, macroscopic serum agglutination tests gave no reaction with *Eberthella typhosa*, *Salmonella paratyphi*, *Salmonella schottmülleri*, *Salmonella hirschfeldii* (paratyphoid C), *Proteus* OXK, *Proteus* OX 19, and *Brucella abortus*.

Investigations for Weil's Disease.—As the patient was already in the twelfth day of his illness, blood culture was not attempted. On January 22, 1937, blood was withdrawn, and the clot, emulsified in saline solution, was inoculated intraperitoneally into a guinea-pig. The guinea-pig did not react with fever, nor did it lose weight, and when it was killed fourteen days after inoculation no evidence of Weil's disease was demonstrated. A saline emulsion of liver and kidney from this guinea-pig was inoculated into a second guinea-pig, which likewise failed to react.

On January 22, 1937, urine was withdrawn, and immediately 8·0 mils of this were inoculated intraperitoneally into a guinea-pig. Dark-ground examination of the centrifuged deposit of urine revealed no leptospiræ. This animal reacted with fever on the third day and continued to do so until the seventh day. On the sixth day heart puncture was performed and 1·0 ml of blood was withdrawn and inoculated into another guinea-pig, which failed to react. Neither of these animals, when killed, gave *post mortem* evidence of leptospirosis.

Agglutination tests performed on serum withdrawn on the twelfth day gave the results shown in Table III.

Comment.

This patient's serum agglutinated conclusively a leptospiral culture of the classical Weil's disease type; also the two cultures isolated from Brisbane rats. "Ballico", a north Queensland strain, and "Pomona", a south Queensland strain, were not agglutinated. It appears that this patient was infected with a strain of leptospira closely allied to, if not identical with, the classical Weil's disease type.

TABLE III.
Agglutination Tests with Serum of R.K.

Strain of Leptospira.	Dilution of Serum.							Control.
	1/10	1/50	1/100	1/500	1/1000	1/5000	1/10000	
Wijnberg	+++	+++	++	+	±	–	–	–
J.J.D.	+++	+++	++	+	±	–	–	–
Rat 101	+++	++	+	+	±	–	–	–
Rat 102	+++	+++	++	+	±	–	–	–
Ballico	±	–	–	–	–	–	–	–
Pomona	–	–	–	–	–	–	–	–
Andaman	lysis +	+	±	–	–	–	–	–
Roesel	+	±	–	–	–	–	–	–
Moscow	+	–	–	–	–	–	–	–
Palembang	–	–	–	–	..	–	–	–
Bangkinang	–	–	–	–	–	–	–	–
Baermann	lysis ±	–	–	–	–	–	–	–

Six days before he became ill this patient left north Queensland, where leptospirosis is endemic. In a review of thirty-seven cases of Weil's disease following a single immersion in water, Schüffner[6] gives the average incubation period as 10·4 days, and 9·8 days for the six fatal cases. Since the patient did not arrive in Brisbane until three days before the onset of his illness, it is certain that he contracted his disease in north Queensland.

Although previously it had not been known that *Leptospira icterohæmorrhagiæ* existed in north Queensland, the occurrence of this case seems strongly to indicate that it does.

Case III.

Clinical History.

The notes in this case are made available by the courtesy of Dr. P. J. Kelly.

J.J.D., a male, aged thirty-two years, had been a sewer worker for two years; during this period he was perfectly well. On February 18 he became suddenly ill, with mild rigors, headache and generalized muscular pain, particularly severe over the chest and abdomen. Next day he felt worse and did not go to work. He was disinclined to eat and was troubled by nausea and dry retching.

On February 22 he was transferred to the Mater Misericordiæ Hospital. The clinical diagnosis at this stage was Weil's disease or typhoid fever. Jaundice began on the sixth day and increased till it was of moderate intensity on the tenth day, when it began to decline slowly. The conjunctivæ were congested, and the patient complained of constant frontal headache of great intensity.

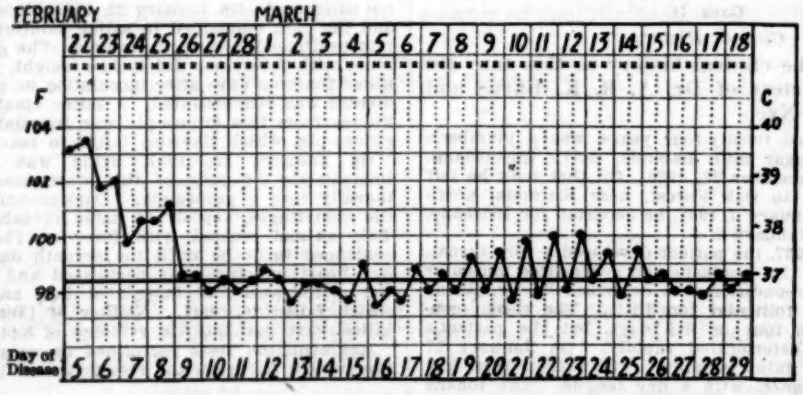

FIGURE I.
Temperature chart of patient J.J.D.

Physical examination on February 22 revealed few positive signs. *Herpes febrilis* was present on the upper lip. There was tenderness in the muscles of the pectoral region and of the abdominal wall, but tenderness was not marked in the muscles of the extremities. The liver and spleen were not enlarged. The urine contained no albumin.

The temperature on admission was 39·7° C. (103·5° F.), and this fell gradually by lysis until it was normal five days later (see chart, Figure I). At the end of the third week of his illness the patient suffered a slight relapse for four days. He had a mild pyrexia with occasional rigors and felt distinctly unwell. However, he apparently made a good recovery, although on the twenty-ninth day of his illness he was still faintly jaundiced.

Pathological Investigations.

On February 22, 1937, blood was cultured for *Eberthella typhosa*. The culture was sterile after five days. (Dr. G. C. Taylor.)

On February 23, 1937, blood was withdrawn and 0·5 ml was inoculated into a tube of Schüffner's medium. After separation of the serum the clot, ground up in sterile saline solution, was inoculated into Schüffner's medium and also into a guinea-pig.

After four day's incubation at 30° C. a growth of leptospiræ was obtained in Schüffner's medium.

The guinea-pig reacted with pyrexia (40·7° C.) on the fourth day and with jaundice on the sixth day. When killed on the tenth day the animal appeared to be recovering. *Post mortem* signs noted were faint jaundice, small intestinal hæmorrhages and typical "butterfly" lungs, but no leptospiræ were seen in the liver, heart, blood or kidney. It was not until the third passage animal had been killed that leptospiræ were recovered from the guinea-pig.

On March 2, 1937, inactive leptospiræ were seen in the urine.

On March 18, 1937, leptospiræ were present in the urine.

Serological Reactions. — Serum withdrawn on the sixth day was tested for leptospiral agglutinins with negative results. Serum withdrawn on the thirteenth day, when tested for agglutinins against various strains of leptospiræ, gave the results shown in Table IV. Serum withdrawn on the twenty-ninth day of disease when tested gave the significant agglutinations shown in Table V.

Comment.

This patient's condition can be regarded as a completely proved case of Weil's disease. A sewer

TABLE IV.
Agglutination Tests with Serum of J.J.D. (12th day).

Strain of Leptospira.	Dilution of Serum. 1/10	1/50	1/100	1/500	1/1000	1/5000	1/10000	1/50000	Control.
Wijnberg	+++	+++	+++	+++	++	+	±	±	–
J.J.D. Culture from patient J.J.D.	+++	+++	+++	+++	++	+	+	+	–
Rat 101	+++	+++	+++	++	+	+	±	–	–
Rat 102	+++	+++	+++	++	+	±	±	–	–
Ballico	++	+	±	–	–	–	–	–	–
Pomona	±	–	–	–	–	–	–	–	–
Andaman	+++	+++	++	++	+	±	±	–	–
Roesel	++	++	+	±	–	–	–	–	–
Moscow	+	+	±	–	–	–	–	–	–
Palembang	+	±	–	–	–	–	–	–	–
Bangkinang	+	±	–	–	–	–	–	–	–
Baermann	+	+	–	–	–	–	–	–	–

TABLE V.
Agglutination Tests with Serum of J.J.D. (29th day).

Strain of Leptospira.	Dilution of Serum. 1/10	1/50	1/100	1/500	1/1000	1/5000	1/10000	1/50000	1/100000	1/500000	Control.
J.J.D.	+++	+++	+++	+++	++	++	+	+	±	–	–
Wijnberg	+++	+++	+++	+++	++	+	±	±	–	–	–
Andaman	+++	+++	+++	++	+	±	±	–	–	–	–

worker by occupation, his illness ran a typical course. Leptospiræ were recovered from the blood by culture and were seen in the urine. The leptospiræ were pathogenic to guinea-pigs.

This case also illustrates the great value of early blood culture in the diagnosis of suspected cases of Weil's disease. Blood withdrawn on the sixth day of illness yielded a growth of leptospiræ in four days. A positive blood culture puts the diagnosis beyond doubt, and the method is advised for all suspected cases if the patients are seen before the seventh day.

The classification of the infecting leptospira is further discussed in another section. Meanwhile, it should be noted that the homologous culture was agglutinated by the patient's serum in a dilution of 1 in 40,000, that maximal agglutination was given by leptospiræ of the classical Weil's disease type, and that leptospiræ of Andaman strain were agglutinated to a significant degree.

Examination of Brisbane Rats, 1936 Series.

During the year 1936, 85 wild rats caught in Brisbane were examined for the presence of leptospiræ. The species and sex of the rats are shown in Table VI.

TABLE VI.
Species and Sex of Rats Examined.

Species.	Males.	Females.	Sex Not Recorded.	Totals.
Rattus norvegicus	22	40		62
Rattus rattus	6	10		16
Species not recorded		1	6	7
Totals	28	51	6	85

The kidney, and in some cases the liver also, was examined microscopically with dark-ground illumination, and then an emulsion of kidney, with, usually, liver and brain also, was injected into guinea-pigs. Each guinea-pig was injected with the organs of one to seven rats. Table VII shows briefly the tests and their results.

TABLE VII.
Examination of Rats and Results.

Test.	Number Examined.	Number Found Infected.
Kidney, microscopic examination	85	2 (Rats 101, 102).
Liver, microscopic examination	26	0.
Kidney, guinea-pig inoculation	74 (into 27 guinea-pigs).	2 (Rats 101, 102).
Kidney, culture	2	2 (Rats 101, 102).

The results of the examinations were entirely negative until December 1. On that day were brought in among others a pair of sewer rats (*Rattus norvegicus*), caught together. Rat 101 was a male of 490 grammes, rat 102 a female of 255 grammes. Active leptospiræ were seen in each kidney of each rat. They could not be detected in any other organs.

Scrapings of kidney from each rat were inoculated into tubes of Schüffner's medium, resulting in each case in a good growth of leptospiræ.

Kidney emulsion from each rat was injected into a guinea-pig. The guinea-pig injected from rat 101 became febrile on the sixth day and was killed on the seventh. It showed petechiæ throughout the lungs, and swollen, congested lymph glands. Leptospiræ were seen in the liver and inguinal glands and were cultivated from the blood. The guinea-pig injected from rat 102 became febrile on the eighth day and was killed when moribund on the eleventh

day. It was deeply jaundiced and showed numerous hæmorrhages in the lungs, intestines and subcutaneous tissues. Leptospiræ were seen in the liver and were cultivated from liver and blood. The infection was passed on from these two into four other guinea-pigs, all of which died with the characteristic picture within seven days.

In contrast to these uniformly fatal results was the behaviour of three guinea-pigs which three or four months previously had had leptospirosis of the Pomona type and which were now injected with the rat 101 strain. All remained apparently healthy. Two became febrile for one day only (the sixth), the other not at all. None of them lost weight or became jaundiced. An attack of Pomona leptospirosis therefore has some protective action against infection with the rat 101 strain, as well as against the Ballico strain.[3]

A small amount of serum was obtained from rat 101 and tested for agglutination with the leptospira obtained from the same rat. The result is given in Table VIII. No serum was obtainable from rat 102.

Table VIII.

Agglutination Test with Serum of Rat 101.[1]

Strain of Leptospira.	Dilution of Serum.			
	1/10	1/50	1/100	1/500
Rat 101	.+++	+	±	–

[1] This test was done at 37° C. for three hours.

Classification of the Strains of Leptospiræ Isolated in Brisbane.

Three strains of leptospiræ were obtained in culture, one from a human patient (J.J.D.) and the other two from rats. In order to reveal their relationships they were tested against various antisera. Some Wijnberg antiserum was kindly sent to us by Dr. Mertens, who also provided the Andaman antiserum. Antisera against the two rat strains were prepared in the laboratory by rabbit inoculation. The antiserum for the human strain was provided by the patient J.J.D. himself. The results of the comparative agglutination tests are given in Tables IV, IX, X and XI.

In the interpretation of these results attention is first directed to the four strains, Wijnberg, J.J.D., rat 101 and rat 102, and their corresponding antisera. The tables show that each of the four antisera agglutinated the three heterologous strains to practically the same extent as the homologous strain.

Furthermore, rat 101 and rat 102 antisera did not agglutinate to a significant degree any other strain of leptospira apart from these four; nor did the Wijnberg antiserum, so far as it was tested. It is clear, therefore, that the two rat strains are identical with each other and with *Leptospira icterohæmorrhagiæ* (Wijnberg).

The human strain, J.J.D., although otherwise behaving similarly to the Wijnberg and rat strains, shows one interesting variation. It has apparently some serological relationship with the Andaman strain. J.J.D.'s serum agglutinated Andaman strain almost as well as it did the homologous strain (Table IV); and Andaman antiserum agglutinated quite definitely a J.J.D. culture, though not to the same degree as it did an Andaman culture (Table XII). J.J.D. strain is the only one of the three Brisbane strains to exhibit this relationship.

The leptospira isolated from the patient J.J.D. is obviously a variant strain and difficult to classify.

Table IX.

Agglutination Tests with Wijnberg Antiserum.

Strain of Leptospira.	Dilution of Serum.								Control.
	1/10	1/50	1/100	1/500	1/1000	1/5000	1/10000	1/50000	
Wijnberg	+++	+++	+++	+++	++	++	+	±	–
J.J.D.	+++	+++	+++	++	+	+	+	±	–
Rat 101	+++	+++	+++	+++	++	+	+	±	–
Rat 102	+++	+++	+++	+++	++	+	±	–	–
Andaman	++	+	–	–	–	–	–	–	–

Table X.

Agglutination Tests with Antiserum "Rat 101".

Strain of Leptospira.	Dilution of Serum.							Control.
	1/10	1/50	1/100	1/500	1/1000	1/5000	1/10000	
Wijnberg	+++	+++	+++	++	+	±	–	–
J.J.D.	+++	+++	++	+	±	±	–	–
Rat 101	+++	+++	+++	+++	+	±	–	–
Rat 102	+++	+++	+++	++	+	±	–	–
Ballico	+	–	–	–	–	–	–	–
Pomona	–	–	–	–	–	–	–	–
Andaman	+	±	–	–	–	–	–	–
Rossel	++	+	±	–	–	–	–	–
Moscow	±	–	–	–	–	–	–	–
Palembang	–	–	–	–	–	–	–	–
Bangkinang	–	–	–	–	–	–	–	–
Baernjann	±	–	–	–	–	–	–	–

TABLE XI.

Agglutination Tests with Antiserum "Rat 102".

Strain of Leptospira.	Dilution of Serum.							Control.
	1/10	1/50	1/100	1/500	1/1000	1/5000	1/10000	
Wijnberg	lysis ++	+++	++	+	+	±	−	−
J.J.D.	+++	+++	++	+	+	±	−	−
Rat 101	+++	+++	+++	++	+	±	−	−
Rat 102	+++	+++	+++	++	+	±	−	−
Ballico	±	−	−	−	−	−	−	−
Pomona	−	−	−	−	−	−	−	−
Andaman	+	−	−	−	−	−	−	−
Rossel	+	±	−	−	−	−	−	−
Moscow	+	−	−	−	−	−	−	−
Palembang	−	−	−	−	−	−	−	−
Bangkinang	−	−	−	−	−	−	−	−
Baermann	±	−	−	−	−	−	−	−

TABLE XII.

Agglutination Tests with Andaman Antiserum.

Strain of Leptospira.	Dilution of Serum.							Control.
	1/10	1/50	1/100	1/500	1/1000	1/5000	1/10000	
Wijnberg	+	−	−	−	−	−	−	−
J.J.D.	++	++	+	±	−	−	−	−
Rat 101	±	−	−	−	−	−	−	−
Rat 102	−	−	−	−	−	−	−	−
Andaman	+++	+++	+++	++	+	+	±	−

However, as its serological properties agree exactly with those of the Wijnberg strain, except for the cross-agglutination with Andaman strain, our present conclusion is to regard it as a somewhat atypical variety of the *Leptospira icterohæmorrhagiæ* type. We hope to study it further.

Another peculiarity of the J.J.D. strain is that it has not proved fatal to the three guinea-pigs so far infected with it. The guinea-pig inoculated with the patient's blood developed pyrexia, jaundice and loss of weight and then began to recover, and two other animals passaged in series have behaved in the same way.

Finally, it is noteworthy that the sera of the first two patients, while giving a diagnostic reaction with Wijnberg strain, gave no significant reaction with Andaman strain. Both patients therefore suffered from Weil's disease of the typical form.

Epidemiological Considerations.

A point of epidemiological interest is that both the first and the third patients were working in the same sewer for some weeks before the onset of their respective illnesses. This suggested that the focus of infection might be a localized one.

The suspected sewer is situated two miles from the place where the two positive rats (rats 101 and 102) were trapped in December, 1936. A series of rats caught in the locality of this sewer was forwarded by the Brisbane City Council to this laboratory during March, 1937. One hundred rats, all *Rattus norvegicus*, were selected for examination; and as a rule the larger rats were chosen. Of these hundred rats, six were found to harbour leptospiræ in their kidneys. Culture was attempted only in two of the six, and guinea-pig inoculation in one. One strain (rat 132) was obtained in pure culture, and the strain inoculated (rat 128) produced typical "butterfly" lungs, but did not kill the guinea-pig. The weights of the positive rats were 445, 237, 530, 194, 172 and 510 grammes.

In 1936 it will be recalled that only two positive rats were found in eighty-five examined (Table VI). In the selected 1937 series six rats were found to be carriers in one hundred examined. These figures show that the carrier rate in Brisbane rats is low compared with that of other cities outside Australia. At present, therefore, only sporadic cases of Weil's disease are likely to occur in Brisbane, and these in the occupational groups most exposed to infection (for example, sewer workers).

Are Imported Rats the Sole Carriers of Weil's Disease?

It is interesting to speculate on another epidemiological aspect of Weil's disease. Two proved strains of *Leptospira icterohæmorrhagiæ* were isolated from an imported species of rat (*Rattus norvegicus*). The other six rats, found to be carriers, were of the same species. In north Queensland, where leptospirosis is endemic, a native rat (*Rattus culmorum*) is the chief carrier of leptospiræ. It is known that more than one type of leptospirosis exists in north Queensland, but we believe that the leptospiræ isolated belong mostly to types other than *Leptospira icterohæmorrhagiæ*. An interesting conjecture

thus arises as to whether *Leptospira icterohæmorrhagiæ* exists in Australia only in imported species of rat, or whether it can, or does, exist in native rodents. Future work will include research to clear up this point, which is important, as it affects the possible spread and control of this severe form of leptospirosis.

Investigation of Sewer.

Visits were made in 1935 and 1937 to the sewer in which the two patients had been working when they became ill. This particular sewer carries storm-water and household drainage. It leads into the Brisbane River and its lower end is flooded with salt-water at high tide. This fact militates against the long survival of pathogenic leptospiræ.[6]

In 1935 slime was scraped from the floor of the sewer, and leptospiræ were seen in this by dark-ground illumination. Two guinea-pigs were injected with the slime, and one guinea-pig was immersed in slimy water for an hour according to the technique of Appelman.[7] One guinea-pig died of peritonitis soon after inoculation, and the other two showed no evidence of leptospirosis. The leptospiræ seen can therefore be regarded as non-pathogenic.

In 1937, rat footprints were noticed in the slime and *débris* of the sewer. Samples of slime and sewer water were brought back to the laboratory. One guinea-pig was inoculated subcutaneously with 1·0 mil of slime, and another guinea-pig was immersed in the water. These animals have so far shown no certain sign of infection.

Summary and Conclusions.

A distinction is drawn between classical Weil's disease, due to *Leptospira icterohæmorrhagiæ*, and other leptospiroses.

Three cases of classical Weil's disease, occurring in Brisbane, are reported. One of the three cases was fatal.

The first patient was a sewer worker who was infected in 1935. His illness was clinically typical, but pathological confirmation of the diagnosis was not at the time forthcoming. Reinvestigation of the case seventeen months later showed that the patient's serum still contained, to a diagnostic titre, agglutinins against *Leptospira icterohæmorrhagiæ*.

The second patient, although he became ill and died in Brisbane, had left north Queensland, where leptospirosis is endemic, six days previously. Serological reactions show that the infecting leptospira was closely allied to, and probably identical with, the leptospira of classical Weil's disease.

The third patient was a sewer worker. His illness was typical, and the diagnosis was confirmed by the isolation of leptospiræ from the blood, by their detection in urine, as well as by agglutination tests.

Both the first and third patients became ill while working in the same sewer, but at an interval of seventeen months.

In a series of eighty-five wild rats examined in Brisbane in 1936, two were found to be carriers of leptospiræ. In a second series of one hundred rats examined in March, 1937, six were found to be carriers of leptospiræ.

The two strains of leptospiræ isolated from rats in 1936 were found to correspond serologically with the Wijnberg strain of *Leptospira icterohæmorrhagiæ*. The human strain isolated, while in general similar to the others, showed some relationship also to an Andaman strain.

This is the first time that cases of classical Weil's disease and the specific type of leptospira have been recorded in Australia.

The presence of *Leptospira icterohæmorrhagiæ* in Brisbane sewer rats, and the accompanying liability of sewer workers to become infected, brings Brisbane into line in this respect with many other cities of the world. The carrier rate is, however, comparatively low, and only sporadic cases of Weil's disease are likely to occur.

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References.

(1) L. S. P. Davidson, R. M. Campbell, H. J. Rae and J. Smith: "Weil's Disease (Leptospirosis)", *The British Medical Journal*, December 22, 1934, page 1137.

(2) J. Smith and L. S. P. Davidson: "The Incidence of Weil's Disease in Fish Workers in Aberdeen", *The Journal of Hygiene*, Volume XXXVI, July, 1936, page 438.

(3) G. E. R. Clayton, E. H. Derrick and R. W. Cilento: "The Presence of Leptospirosis of a Mild Type (Seven-Day Fever) in Queensland", THE MEDICAL JOURNAL OF AUSTRALIA, May 1, 1937, page 647.

(4) S. Postmus: Quoted by N. Hamilton Fairley (5).

(5) N. Hamilton Fairley: "Weil's Disease among London Sewer Workers", *The British Medical Journal*, July 7, 1934, page 10.

(6) W. A. P. Schüffner: "Recent Work on Leptospirosis", *Transactions of the Royal Society of Tropical Medicine and Hygiene*, Volume XXVIII, June, 1934, page 7.

(7) J. M. Appelman: "Isolation of Leptospira Icterohæmorrhagiæ from Water", thesis for the Doctorate of Medicine of Leiden University, abstracted in *The Tropical Diseases Bulletin*, Volume XXXI, July, 1934, page 514.

CONTEMPORARY NEURO-SURGICAL PRACTICE.

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THE following considerations of recent methods of neuro-surgical treatment are based on observations made by the writer during a visit to America in the early months of this year as a guest of the Rockefeller Foundation. The conditions to be discussed here have suggested themselves for consideration for one or more of three reasons, namely: the method of treatment described may only lately have been developed, or it may have been recently modified, or the condition amenable to recognized treatment may only recently have been discovered to occur with considerable frequency and to be the constant cause of symptoms with which it was not previously classically associated.

Some of the statements in this article have been personal communications made to the writer, and a number of these are based on investigations of clinical material the results of which have not yet been published. On the other hand, some of the procedures described here may be familiar, particularly to those who are interested in the surgery of the nervous system. These, however, have been included, since they may be of considerable general interest. The following conditions will be discussed chiefly from the points of view of recognition and treatment: (i) sciatic pain, (ii) autonomic pain, (iii) essential hypertension, (iv) Raynaud's disease, (v) facial nerve grafts, (vi) Ménière's disease.

Sciatic Pain.

In cases in which pain is present in the distribution of one or both sciatic nerves, search should always be made for herniation of the *nucleus pulposus* of one of the intervertebral disks. Recognized ten years ago, it has only recently become known that this condition is one of the most common causes of sciatic pain. Until 1936 only eleven patients suffering from herniated disk had been operated on at the Mayo Clinic, the *nucleus pulposus* being removed, while in the past eighteen months fifty-six such patients have been operated on there and cured. Horrax and Poppen had five patients with post-operative herniated disks in the Deaconess Hospital in Boston at one time. In another instance seventeen patients had been operated on in one clinic in three months. I witnessed eight removals of herniated *nuclei pulposi* in the month of February. The condition, therefore, is quite common, and the recognition of this fact is important in the investigation of compensation and industrial cases in which the patient complains of low lumbar and sciatic pain. Pathologically the condition was originally thought to be neoplastic and was described either as a fibroma or enchondroma of the intervertebral disk. Adson removed a herniated disk in 1924, which was recorded at the time as an enchondroma. When herniation takes place the *nucleus pulposus* is squeezed posteriorly and forms a definite tumour under the posterior longitudinal ligament on the posterior aspect of the vertebral bodies. It usually comes to lie on one side or the other of the midline, and since in 80% of cases herniation takes place between the fourth and fifth lumbar vertebræ, pressure is exerted on the roots of the *cauda equina* at that level. In one group of 56 cases, 41 were lumbar, eight thoracic and six cervical. Multiple herniations have been recorded. Trauma is presumed to be the main ætiological factor, but in many cases no history of injury can be elicited. Sciatic pain is a constant symptom in all the lumbar cases. It is usually unilateral, may be bilateral, and may be associated with low back pain. Paræsthesiæ are rare, but have been reported. In lumbar cases sciatic pain is usually the only symptom, and there may be no signs other than depression of one or both Achilles jerks.

In the thoracic cases pain is unusual and paraplegia is more common. Diagnosis is confirmed by examination on a tilt table after injection of five cubic centimetres of lipiodol into the lumbar subarachnoid sac. Definite notching of the lipiodol may be seen at the level of herniation. The cerebro-spinal fluid protein is as a rule but slightly increased. The operation offers some technical difficulties, but if notching of the lipiodol at a constant level can be demonstrated, the patient can be assured of post-operative relief of the pain.

Subarachnoid Alcohol Injection.—Those cases of sciatic pain for which no organic lesion can be detected have been assumed to be due to a trunkulitis or radiculitis of sciatic nerve fibres. Various procedures, such as epidural and intraneural injections of normal saline and "Novocain" solutions combined with physical therapy, have been applied to these cases with indifferent success. The most recent method of treating such pain is by the injection into the lumbar subarachnoid space of 90% alcohol. The injection is made with the patient in the prone position and the pelvis rotated to one side or the other, so that the sensory roots on the same side as the pain may be destroyed selectively. Owing to the much lower specific gravity of the alcohol (the specific gravity of alcohol is 794 and of cerebro-spinal fluid 1006), it does not mix with the cerebro-spinal fluid, if carefully injected, but floats on the surface to encircle the dorsal roots of the *cauda equina*. A glass syringe with a threaded stem should be used to permit minim by minim injection only and to prevent sudden injection of any larger quantity. The patient should remain in the prone position for thirty minutes after injection. If the operation is thus carefully performed, there is no risk of disaster and the procedure is not followed by bladder disturbances or changes in reflex action or motor power. If pain is not completely relieved by the first injection, a second one may be given, the same precautions being

used. Spinal subarachnoid alcohol injection is now replacing the more tedious and difficult operation of lumbar chordotomy in all cases of intractable pain transmitted centrally from pelvic or peripheral structures in the dorsal roots of the *cauda equina*. Although these injections have been successfully performed in the thoracic and cervical regions, they would seem to be attended by considerable risk of spinal cord trauma at those levels.

Autonomic Pain.

It is not proposed to consider under the heading of autonomic pain the Learmonth operation of presacral neurectomy for pain arising in uterine and vesical disease. This operation has achieved a vogue with gynæcologists for dysmenorrhœa and chronic interstitial cystitis. Its use in males is limited, since it is succeeded by sterility, but in females it has proved to be an eminently successful operation for relieving pain in cases of pelvic visceral disease.

Jason Mixter and J. C. White, at the Massachusetts General Hospital, have been interested in the treatment of cardiac pain in *angina pectoris*. They have treated 50 patients suffering from severe *angina pectoris* by alcohol injection of the upper three thoracic sympathetic ganglia. This injection destroys the visceral afferent nerve fibres from the heart which pass through these ganglia, and in all the cases in which injection has been carried out at the Massachusetts General Hospital, complete or almost complete relief from anginal pain has resulted. The technique of injection is relatively simple and injection is performed under local anæsthesia. In a number of cases neuritic pain in the first, second and third thoracic dermatomes succeeds the injection owing to irritation of the corresponding thoracic nerves. This neuritic pain, when it occurs, usually passes off in a week or two, but it may persist for longer. It is usually of low intensity and quite tolerable to patients who have experienced anginal attacks. J. C. White told the writer that in only one case was the injection followed by such severe neuritic pain that the patient said he would have preferred not to have had the injection.

Several patients injected five years ago had had complete freedom from pain for the entire period. As might be expected from recent observations on the physiology of referred pain,[1] pain referred to the second to the fourth thoracic dermatomes in angina is abolished by this injection in addition to the precordial pain of visceral origin.

The objection has been levelled at this operation that it may submit the patient to cardiac overload and thus curtail this survival period, since by abolition of pain, warning of cardiac distress is no longer present. J. C. White[2] says, however, that after injection, although these patients may not suffer pain of any consequence they are still warned of attacks by palpitation, by a sense of precordial constriction, or by slight painful sensations on the right side. The procedure is therefore not dangerous for this reason.

The same operation has also been employed with rather less success in causalgias of the superior extremities and in migraine. In the latter disorder the efficacy of visceral afferent interruption either by alcohol injection or sympathectomy may be estimated by the response to ergotamine tartrate. If a migrainous headache responds to the intravenous injection of 0·25 milligramme of ergotamine tartrate, it will often also respond to autonomic denervation. In migraine, if pain is the only symptom and no motor or sensory migrainous equivalents occur, it may be adequate only to divide the afferent sympathetic plexus on the middle meningeal artery, a procedure which can be carried out under local anæsthesia. Paravertebral sympathetic injection has also been advocated for pain occurring in a number of conditions, such as scleroderma, *thrombo-angiitis obliterans*, intermittent claudication and chronic arthritic disease. The efficacy of the injection to relieve pain may be estimated by injection of "Novocain" solution before the alcohol.

Hypertension.

A number of operations are being practised at the present time for the treatment of the symptoms of vascular hypertension. All of these aim at autonomic denervation of the splanchnic area with or without subtotal adrenalectomy. This may be done in one of several ways. Heuer and Page have treated a series of patients by laminectomy and bilateral section of the motor roots of the cord from the fifth thoracic to the second lumbar segment inclusive. This divides all preganglionic fibres to the splanchnic area, and from the reports of these authors would appear to be an effective treatment for hypertensive symptoms. It is, however, a very extensive operation, produces great weakness of the back, paralyses the abdominal muscles, requires that a surgical corset be worn subsequently and is a debilitating procedure in any person who does not follow a completely sedentary occupation. Crile[3] has reported his results after resecting the cœliac ganglia and denervating the aortic plexus in 106 cases of essential hypertenesion.

At the Mayo Clinic, Adson and Craig[4] are now operating on hypertensive patients in a two-stage operation by the abdominal route, removing the abdominal sympathetic chains on the two sides, the splanchnic nerves on the two sides, and in some cases removing the upper third of each suprarenal gland. Physiologically the partial adrenalectomy would not appear to be of much value, since it has been shown experimentally that the remaining portion of the gland subsequently undergoes hypertrophy. This operation is a very thorough autonomic denervation, but in its results does not appear to be any more effective than the operation recommended by Max Peet[5] at Ann Arbor, which has the great advantage from the patient's point of view that it is carried out at one sitting. Peet removes portion of the eleventh rib on each side and through the spaces thus provided resects the splanchnic nerve on each side and the ninth, tenth, eleventh and twelfth thoracic sympathetic ganglia with their

medially directed branches into the aortic sympathetic plexus. Peet, who is an extremely facile operator, does the entire operation in about forty-five minutes.

The symptoms for which operation is recommended are headaches, dizziness, dyspnœa, insomnia, nocturia and failing vision, and the following criteria should be fulfilled before operation is undertaken. The patient should not be over fifty years of age, and systolic blood pressure readings as high as 200 millimetres should have been observed. The specific gravity of the urine should not be below 1010 and the blood non-protein nitrogen should not be above a high normal level. Such changes in the fundi as papillœdema, retinal hæmorrhages and exudates are not contraindications to operation, and indeed some brilliant results have been obtained in patients exhibiting these signs.

Peet's series of 120 patients have been studied post-operatively for periods as long as two years. Considerable symptomatic improvement was noted in 77% of cases; 15% of patients were classified as completely cured, since the blood pressure had remained at a normal level for longer than six months and there had been complete relief from symptoms. Another 33% were able to resume their normal activities when before operation their symptoms were practically incapacitating. A further 29% exhibited a very considerable relief from symptoms, with a drop in systolic pressure from 25 to 50 millimetres of mercury. Eight patients had had strokes before operation. None have had strokes subsequently.

It would be of little value to discuss here the theories of the ætiology of essential hypertension. It may be said, however, that there is good evidence to suggest that it is a neurogenic disorder produced by some pathological condition of the autonomic centres of the diencephalon. The work of Richards and Goldblatt has shown that excessive splanchnic discharge will cause chronic hypertension, and that this effect is due to alteration of renal function by vaso-constriction is supported by their observation that hyperpiesia is produced in experimental animals by graded constriction of the renal arteries. Govaerts and Dicker[6] have also shown that hypertensive substances are formed in the kidneys when the blood supply is reduced by ligating or clamping the renal vessels. On the other hand, it is known that hypertension does not occur after bilateral nephrectomy, although the animal may live for two weeks or more after operation. It would seem, then, that depreciation of renal blood supply is an important causal factor in the production of essential hypertension, but that the latter does not occur when both kidneys are completely excluded from the general circulation. Peet believes that post-operative dilatation of renal vessels is the basis of his favourable surgical results. It may no longer be contended logically that hypertension is a compensatory change to ensure adequate renal or cerebral blood supply since a number of patients with a pre-operative systolic pressure of 250 millimetres of mercury or more have for periods of six months to two years after operation remained in perfect health, with systolic pressures of 140 millimetres of mercury or less. In only one instance has any cerebral disturbance been reported which might be attributed to the operation. In that case the patient exhibited a weakness of one arm, which passed off after a few hours.

There would appear to be a strong hereditary factor in essential hypertension. Recent studies suggest that some 18% of the adult population in America shows the hypertensive type of reflex dermal temperature change when one extremity is plunged into cold water.[7] About the same percentage of school children show hypertensive dermal reflex responses. Hines, at the Mayo Clinic,[8] has followed up the inheritance aspect of essential hypertension by studying in Minnesota a large number of four-generation families, with as many as ninety individuals in each, and is accumulating results which suggest the possibility of some Mendelian transference of essential hypertensive disease.

It would seem that there is an extensive field for investigation and classification of hypertensive cases. Allen, of the Mayo Clinic, is concerned with this problem at present, and physicians generally are studying hypertensive patients with fresh enthusiasm so that some classification may be made by which the value of operation may be estimated in particular cases. Frequently there may not be any general vascular spasticity in hyperpiesia, so that the intensity of vascular spasm, measured by heating or cooling one limb or the whole body, by intravenous injections of adrenaline or vaccines or by study of the blood pressure fall after administration of "Avertin", does not provide consistently reliable prognostic information. It still remains for some experimental test or clinical classification to determine whether particular patients require operative or palliative treatment.

One fact was observed by the writer in studying a number of these cases, namely, there does not appear to be any quantitative relation between the relief of symptoms and the post-operative fall of blood pressure. Immediately after section of the splanchnic nerves blood pressure falls in every case by 25, 50 or more millimetres of mercury. In the subsequent two weeks the pressure may remain at the new low level or it may gradually rise to some point closer to the pre-operation level. In patients with a 30-millimetre drop after operation the relief of symptoms may be as great as or even greater than it is in those with a 60-millimetre drop. Even though splanchnicectomy produces a variable fall in blood pressure, it always appears to stabilize the systolic pressure, and therein may perhaps be found the value of the operation. Study of a number of records of blood pressure after splanchnicectomy, when the patient is at rest, discloses a regular level varying only by 5 to 15 millimetres of mercury. This may be contrasted with similar resting pre-operative records showing sharp peaks and falls

from day to day and hour to hour. It is fortunate that this surgical procedure has been fostered by men of the standing of Peet and Adson, and that as the result of their intensive studies and conservative attitude it has not been discredited by wholesale splanchnicectomy in unsuitable cases.

Raynaud's Disease and other Vasospastic Conditions.

Recent advances in the understanding of the physiology of the sympathetic nervous system have introduced a new principle into the surgical treatment of diseases caused by vascular spasm. The surgical application of this principle has arisen largely from the clinical and experimental studies of White, Smithwick and their colleagues[9] at the Massachusetts General Hospital. These workers became interested in this aspect of sympathetic surgery as a result of the discrepancy between the after-effects of operation in patients with Raynaud's disease in the upper extremities and in the lower extremities respectively.

Resection of the second and third lumbar sympathetic ganglia is now generally agreed to be an adequate surgical procedure for restoring the circulation in the lower limbs in favourable early cases of Raynaud's disease. In the upper limbs, on the other hand, the most extensive surgical procedure, by whatever route and following with scrupulous care the particular technique which has been described by so many different surgeons (which it is not proposed to review here), although it may often produce early relief from pain, coldness and spasm, nevertheless has been consistently disappointing in its late results. Ross,[10] reviewing the results of sympathectomy in cases of Raynaud's disease in the upper extremities, observed definite improvement in the limb circulation during the first few weeks after operation, but remarked that subsequently the patients have wished that their hands had remained as warm as they were soon after operation.

The possible explanation of these unsatisfactory results arises from the principle that smooth muscle soon after denervation becomes hypersensitive to adrenaline circulating in the blood stream. It has been known since 1904[11] that the denervated *dilator pupillæ* muscle will contract in response to a dose of adrenaline which will not affect the normal iris. Cannon and his co-workers[12] observed that after sympathetic denervation the heart became hypersensitive to adrenaline in the blood stream and that sympathectomy did not prevent cardiac acceleration until the suprarenal glands had been removed or denervated. So sensitive is sympathectomized smooth muscle that in a cat in which the cervical, thoracic and lumbar sympathetic chains have been removed on both sides, the minute amount of adrenaline which is liberated as a chemical transmitter of impulses to the plain muscle of the tail hairs, when the post-ganglionic fibres are stimulated electrically, is sufficient to produce a marked tachycardia. The completely denervated heart has been found to be sensitive to one part of adrenaline in fourteen hundred million.

It has been found[13] that this same principle applies to smooth muscle in the walls of blood vessels; the rabbit's ear, for instance, after superior cervical ganglionectomy will cool almost to room temperature during the secretion of adrenaline which takes place in hypoglycæmia caused by insulin. The same authors demonstrated a fall of skin temperature of approximately 14° F. produced by stimulation of the suprarenal glands by insulin-induced hypoglycæmia (30 units of insulin) in the fingers of patients after cervico-thoracic sympathectomy. In another patient an injection of the same small dose of adrenaline produced a fall of skin temperature of approximately 1° F. in the toes sixteen months after lumbar sympathectomy, and a fall of 8° F. in the fingers six months after cervico-thoracic sympathectomy. It must be admitted from this and from the results of many other experiments that by degeneration of its post-ganglionic autonomic innervation smooth muscle in blood vessels becomes sensitized to the presence of sympathomimetic hormones circulating in the blood stream. This fact would seem adequately to explain the indifferent results of sympathetic ganglionectomy for Raynaud's disease and other vaso-spastic conditions in the superior extremities; while, on the other hand, the uniformly good results of lumbar ganglionectomy in the same diseases in the inferior extremity would appear undoubtedly to depend upon the fact that after removal of the second and third lumbar sympathetic ganglia, all pre-ganglionic fibres to the inferior extremities are interrupted, but the great bulk of the post-ganglionic sympathetic supply, arising from cells in the fourth lumbar to third sacral ganglia inclusively, is still intact. The integrity of this innervation prevents the development of smooth muscle sensitization and consequently the return of pain, coldness and spasm.

A particularly interesting phenomenon which would appear to be directly related to adrenaline sensitization succeeding post-ganglionic denervation has been described recently by Govaerts.[14] He observed that the persistent vaso-constrictor discharges in post-ganglionic sympathetic fibres, which were first described by Adrian, Bronk and the present writer,[15] cease after section of the pre-ganglionic fibres. He observed, however, that within a few weeks persistent spontaneous discharge was initiated from the intact post-ganglionic cells. The question at once arises: "Is this spontaneous discharge the factor preventing adrenaline sensitization of the effector at the periphery?" In any case, to relieve vasospasm it would appear essential not to interfere with post-ganglionic innervation, but only to section the connexions between the central nervous system and the para-vertebral sympathetic ganglia. Recognizing the differential results of pre-ganglionic and post-ganglionic nerve section, Smithwick has suggested a new operation for Raynaud's disease and other vasospastic conditions in the upper extremities. In this operation the inner inch of the third rib and the transverse process of the third dorsal vertebra

are resected. The proximal portions of the second and third intercostal nerves are resected from the intervertebral foramina outward, the dorsal sympathetic trunk is sectioned just below the third dorsal ganglia, and the upper end of the second trunk is brought out of the thorax and sutured without tension to the muscles of the back. This procedure removes all the pre-ganglionic fibres to the superior extremity with the exception of the small number which pass out as pre-ganglionic arm innervation to the first dorsal ganglion. This modified type of sympathectomy is not followed by a recurrence of vascular spasm due to the action of adrenaline on the blood vessels in the sympathectomized area.

Smithwick has reported a series of such operations on thirty-three upper extremities in twenty-three patients. All his patients have shown surface temperature rises after operation ranging between 5° and 20° F. at the tips of the fingers. He states:

> The temperature rise is dependent upon the amount of organic occlusion already present. In all but one instance the hands appear to be completely sympathectomized, being pink in colour and dry.

Telford[16] has also devised an operation based on the same principle, using an anterior approach. After these operations no clinical evidence of the recurrence of vascular spasm has been noted.

It should be remembered that in the operations described for the treatment of essential hypertension only the pre-ganglionic fibres are severed unless the cœliac and other collateral sympathetic abdominal ganglia are removed. With these ganglia and their peripheral nervous connexions intact, all the blood vessels in the kidneys and in the intestine still retain their post-ganglionic innervation.

Facial Nerve Grafts.

The method of nerve grafting developed by Ballance[17] and Duel[18] has been used for restoring the continuity of the facial nerve after its interruption by mastoid disease or operation. This delicate and interesting operation is now being done chiefly by Tickle, of the Manhattan Ear, Nose and Throat Hospital, who recently crossed to England to operate on two patients. Facial palsy succeeding lesions of the facial nerve in the temporal bone may be relieved by spino-facial or hypoglosso-facial anastomosis. These simple neuro-surgical procedures are open to several objections. In the first place dissociated facial movements are not restored. The resting posture may be normal, but voluntary movements tend to involve the whole of the affected side of the face. Emotional expression is often altered and in women this alteration may prove displeasing. Paralysis of the trapezius is a real disability for those engaged in manual occupations, and the spinal accessory nerve should therefore not be used for anastomosis with the facial in persons engaged in laborious occupations. In some cases, too, a troublesome dysarthria may succeed hypoglosso-facial anastomosis.

Facial nerve grafting in the temporal bone is free from these objections. The operation is tedious, as the injured nerve must be exposed in the bone from the stylo-mastoid foramen even up as far as the geniculate ganglion. The ragged ends are squared off with sharp scissors and the length of the missing section is measured. The anterior cutaneous nerve of the thigh is exposed and an adequate length of nerve is excised and laid in the facial canal with its ends exactly approximated to the central and peripheral ends of the facial nerve. No sutures are required, as the graft is kept securely in place by capillarity. There is no polarity of conduction imposed on the graft, or, in other words, it makes no difference whether the graft is placed *in situ* in its original conduction-direction or is reversed. Nor does it matter whether the graft is prepared or not by allowing the fibres to degenerate after a first-stage operation. The essential feature in restoring continuity and function in the nerve appears to be the introduction of a chain of neurilemma cells into the gap. Regeneration then takes place. Tickle now uses all fresh grafts and has done this operation in 150 cases with restoration of normal facial nerve function in over 90%. The only contraindication to this operation is the absence of facial movements on galvanic stimulation of the facial musculature. According to Tickle, the presence of infection does not interfere with regeneration.

Ménière's Symptom Complex.

The condition of paroxysmal aural vertigo known as Ménière's disease appears to be much more common, at least in America, than might be expected. During the writer's recent visit to the Johns Hopkins Hospital, five cases were seen in Dr. Walter Dandy's clinic in one week. Clinically, the condition has been clarified in the last few years, owing perhaps mainly to pre-operative neurological studies. Ménière's name continues to be associated with this condition, although its pathological basis is not considered to be that put forward by him in his original description in 1861. The syndrome is clear-cut and consists of paroxysmal attacks of vertigo in patients with impaired cochlear function on one or both sides. Deafness is usually more marked on the side to which the symptoms are referred. Nausea, vomiting and spontaneous nystagmus may occur in the attacks, sometimes with tinnitus, and usually the caloric reaction is absent on the affected side. This is a completely debilitating condition, which, fortunately, has responded particularly well to surgical treatment.

Its pathological basis is still in question; some hold that the disease originates in the internal ear, while others believe that the auditory nerve or the vestibular nucleus is the site of the lesion. However, in some cases in which histological *post mortem* examination has been made, these structures have appeared to be perfectly normal. The operation which has been devised for the relief of this condition is that of section of the eighth nerve. If the condition is definitely unilateral there can be no uncertainty as to which eighth nerve should be sectioned, but in the event of a bilateral symptomatology the nerve should be sectioned on the side on

which audiometer tests demonstrate the greater depreciation of cochlear function. This is the case even if the caloric reaction is present on that side and absent on the contralateral side. If there is considerable depreciation of cochlear function, both the vestibular and cochlear fibres may be sectioned; but in the presence of only slight deafness, the vestibular fibres are cut and the cochlear fibres are left intact. The operation is a relatively simple neuro-surgical procedure, and in capable hands is attended by no mortality. Transient diplopia occasionally succeeds vestibular nerve section, but lasts only for a few days. Spontaneous nystagmus may also appear for a few hours after the operation. The patient is normally discharged from hospital within a week and can confidently resume his occupation without fear of further vertiginous attacks.

If the condition is bilateral both vestibular nerves may be sectioned without the production of incapacitating equilibratory disturbances. When this is done the patient may be a little unsteady in the Romberg position, particularly when the eyes are closed, but the degree of instability is not sufficient to interfere with ordinary activities.

A considerable advance has recently been made in the treatment of this condition as the result of the work of Dr. Furstenberg[19] and his colleagues at Ann Arbor. Dr. Furstenberg concluded from the experiments that the symptoms of Ménière's disease were due to the retention of sodium by the body and corresponding water retention if an adequate supply was available. He believes that in Ménière's disease the local tissues involved have either an increased avidity for sodium or an unusual sensitivity to it. As the result of his experimental work he has introduced a dietetic treatment for Ménière's syndrome and has reported the successful treatment of fourteen typical cases. The writer received favourable reports of this method of treatment in many clinics and was even informed by one neurological surgeon that he no longer operated in these cases, but used Furstenberg's diet. Furstenberg states that the therapeutic indications are: (i) to permit as small an intake of sodium as possible, and (ii) to prevent the accumulation of sodium by the body.

The former is attained by diet and the latter by the administration of ammonium chloride. If these two factors are adequately controlled, the intake of water does not need to be considered. Furstenberg's argument appears to be a good one, since not only has he succeeded in obtaining complete relief from vertiginous attacks by this method, but he has also produced the attacks in every case by the administration of sodium. As this method appears to be so effective, the following summary of treatment is reproduced here from one of Furstenberg's articles:

1. Protein, unrestricted or forced.

2. Calories, as indicated.

3. Medication. Ammonium chloride, 3·0 grammes, with each meal, in capsules (six capsules, each containing seven and a half grains, taken during the meal), three days on and two days off. The capsules should not be replaced by the chocolate-coated or the enteric-coated pills, because the latter sometimes pass through the gastro-intestinal tract without being absorbed. The ammonium chloride can be given in this dosage for an indefinite time without injurious effects. We have had patients with nephritis receiving ammonium chloride in this manner for a period of five years.

4. Water intake unrestricted, although excessive quantities of liquids should not be taken.

5. Diet, approximately neutral, low sodium diet.

Group *A*. The following foods may be taken daily:

1. Eggs, meat, fish and fowl as desired.

2. Bread as desired.

3. Cereal, one of the following: farina, oatmeal, rice, puffed rice or wheat.

4. Potato and one or more servings of any of the following: (*a*) macaroni, (*b*) spaghetti, (*c*) rice, (*d*) corn, (*e*) cranberries, (*f*) prunes, (*g*) plums.

5. Milk as desired.

6. Vegetables and fruits daily of any fruit and of any vegetable not included in Groups *B* and *C* as desired.

7. Butter, cream, honey, jellies, jams, sugar and candy permitted as desired.

8. Tea and coffee as desired.

Foods to be Avoided.

Group *B*: All salt meats and fish, or bread, crackers and butter prepared with salt; carrots, clams, condensed milk, raisins, caviare, cowpeas, olives, spinach, cheese, endive, oysters.

Foods to be Taken no more than Twice a Week.

Group *C*: Lima beans, beets, buttermilk, cantaloupe, cauliflower, celery, chard, dried coconut, dried currants, dates, figs, horseradish, kohlrabi, limes, muskmelon, peanuts, peaches, mustard, pumpkin, radishes, rutabagas, strawberries, turnips, turnip tops, watercress.

Note: All food must be prepared and served without salt.

References.

(1) Gilbert Phillips: "Recent Observations on Referred Pain", *Australian and New Zealand Journal of Surgery*, Volume VI, No. 4, April, 1937, page 350.

(2) J. C. White: Personal communication, February, 1937.

(3) G. Crile: "Surgical Treatment of Essential Hypertension: Report of Progress in 106 Cases", *Cleveland Clinical Quarterly Journal*, Volume III, July, 1936, page 201.

(4) A. W. Adson: "Essential Hypertension: 1. General Considerations", "Essential Hypertension: II. The Rationale and Method of Surgical Treatment", *Proceedings of the Mayo Clinic*, Volume XII, Number 1, January 6, 1937, and Volume XII, Number 4, January 27, 1937.

(5) M. M. Peet: "The Surgical Treatment of Hypertension", *University Hospital Bulletin of the University of Michigan, Ann Arbor*, Volume III, January, 1937, pages 1-4.

(6) P. Govaerts and E. Dicker: "*Les substances vasopressives présentes dans le sérum des néphrétiques hypertendus existent-elles dans le sérum des sujets normaux?*" *Comptes rendus des séances de la société de biologie*, Volume CXXII, 1936, page 809.

(7) E. V. Allen: Personal communication, February, 1937.

(8) E. A. Hines, junior, and G. E. Brown: "The Hereditary Factor in the Reaction of Blood Pressure to a Standard Stimulus (Cold): Primary Report", *Proceedings of the Mayo Clinic*, Volume X, Number 24, June 12, 1935.

(9) R. H. Smithwick: "Modified Dorsal Sympathectomy for Vascular Spasm (Raynaud's Disease) of the Upper Extremity", *Annals of Surgery*, Volume CIV, Number 3, September, 1936.

(10) J. P. Ross: "Results of Sympathectomy: Analysis of Cases Reported by Fellows of Association of Surgeons", *The British Journal of Surgery*, Volume XXIII, October, 1935, page 433.

(11) S. J. Meltzer and C. M. Auer: "Studies on the Paradoxical 'Pupil Dilation Caused by Adrenalin'. No. 1. The Effect of Subcutaneous Injections and Instillations of Adrenalin upon the Pupils of Rabbits", *American Journal of Physiology*, Volume XI, 1904, page 28.

(12) W. B. Cannon *et alii*: "Studies on Conditions of Activity in Endocrine Glands: Lasting Preparation of Denervated Heart for Detecting Internal Secretion with Evidence for Accessory Accelerator Fibres from Thoracic Sympathetic Chain", *American Journal of Physiology*, Volume LXXVII, 1926, page 326.

(13) J. C. White: "The Autonomic Nervous System", 1935.

(14) P. Govaerts: *Archives internationales de médecine expérimentale*, October, 1936.

(15) E. D. Adrian, D. W. Bronk and Gilbert Phillips: "Discharges in Mammalian Sympathetic Nerves", *Journal of Physiology*, Volume LXXIV, Number 2, February 8, 1932.

[39] E. C. Telford: "Technique of Sympathectomy", *The British Journal of Surgery*, Volume XXIII, October, 1935, page 448.
[40] C. Ballance: "Operative Treatment of Facial Palsy, with Observations on Prepared Nerve-Graft and on Facial Spasm", *Proceedings of the Royal Society of Medicine*, Volume XXVII, August, 1934, page 1367.
[41] A. B. Duel: "Surgical Treatment of Facial Nerve Paralysis", *Acta Oto-laryngologica*, Volume XXII, 1935, page 373.
[42] A. C. Furstenberg *et alii*: "Ménière's Symptom Complex: Medical Treatment", *Annals of Otology, Rhinology, Laryngology*, Volume XXXIV, Number 4, December, 1934, page 1035.

HEPATIC NECROSIS.

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DISTURBANCES of the liver are notoriously difficult of diagnosis in their early stages or if mild in degree. When jaundice occurs it is probable that there is liver disease, but in many of the cases to which I shall refer there is no apparent jaundice in the early stages, and often only a mild "retentive" jaundice throughout the greater part of the course of the illness. These cases have been described during the last twenty years under the name "hepatic necrosis", and cases seem to have been recurring throughout the world with increasing frequency. Although this is a pathological condition with a number of primary causes, the changes in the liver are similar in all cases and the symptomatology and results vary only with the degree of necrosis and subsequent changes, and with the incidence of the cause. It was well known among the Greeks and Romans and probably among most of the Eastern and Mediterranean peoples, and during the Great War it became increasingly frequent among troops on the Eastern Front. It was then described as "camp jaundice", "infective jaundice", in more chronic cases "infective jaundice with cirrhosis", and finally "necrosis of the liver". At present it is admitted to our text-books in the one form of Weil's disease, but the common forms of "camp jaundice" are certainly not due to the *Leptospira icterohæmorrhagiæ*.

If cases can be recognized and properly investigated in an early stage it may be possible to determine some specific infective agent as the cause of the more common types of the condition.

We have first to discard much of the teaching of our text-books. It has been in the by-ways of medical literature that the truth has cropped out, often not fully recognized by the authors and generally unappreciated by readers. Accordingly, I must briefly review some historical facts that are generally overlooked. To the Greeks and Romans the appearances of the liver in health and disease were well known from the naked eye aspect; apparently they met with many more cases of the acute or subacute variety that can be justly called "necrosis of the liver" than of the more chronic types to which writers of other nations, perhaps outside tropical or semi-tropical zones, applied the name "cirrhosis"; for the word "cirrhosis" is derived from the word κιρρός, which signifies "tawny yellow" or "tawny red", exactly describing the appearance of the liver in subacute and acute cases.

In England it was laid down that all cirrhosis with nodular changes was due to the ingestion of alcohol; it was suggested that hypertrophic biliary cirrhosis might be due to other poisons. As I was interested in the diseases of children, it soon became clear to me that cirrhosis in children could not often be due to alcohol, especially in Australia, where children are rarely given alcohol in any form. Sir Thomas Barlow, in 1884, had published a case of a child who had been given gin daily practically from birth; later, Ernest Jones published a series of seventy-five cases from the literature in all of which the children had been given alcohol at some time. This may well have been at a time when beer was as universal a drink as tea is with us, it being common for a mother to take a two or three years old child into a bar for a "beer and cheese". But Byrom Bramwell, in 1898, published a case of a boy of nine years of age who died of nodular cirrhosis after an actual illness of only four weeks and whose three sisters had previously died in a similar manner; in none of these was there any possibility of an alcoholic cause. Such cases were published again and again, and then vague toxins, vinegar, condiments, and even medicinal tinctures were incriminated. Still the term "nodular or alcoholic cirrhosis" is, today, retained in our text-books.

Hale White, in 1903, suggested that it was not the alcohol itself that caused cirrhosis, but some poison, such as arsenic, that was the cause, the fore-runner of our toxic necrosis of the liver, recognized when we began to treat syphilis with arsenical compounds. In 1902 I met with my first puzzling case, that of a Chinese, with the clinical picture of cirrhosis of the liver and death after only a short illness. On autopsy patches of yellowish-red necrotic tissue with bands of fibrosis were found throughout the liver.

Investigations of liver conditions over the last twenty or thirty years have greatly increased our knowledge of liver disease, and of these I would particularly mention Cantlie's observations on the anatomy of the circulatory system of the liver in 1898, followed by observations in the same direction by Barcroft (1912) and Dale (1932). The discovery by Aschoff (1911) of the reticulo-endothelial tissue, and the realization that the cells of that tissue are instrumental in the formation of bilirubin have clarified our knowledge, and the painstaking work of McNee, which, I believe, he began in 1911, and which he is still continuing, is adding to it still further.

It is necessary to commence our study among children if we are to get a clear view of the subject, and it is my general opinion that the divorce of adult medicine from the study of disease in children is responsible for much of the chaos that prevails. The search for possible causation is much more limited, the pathological processes are much more rapid in their evolution, and the possibility of partial or complete recovery is much greater in children.

Icterus Neonatorum.

Beginning in childhood, we are immediately faced with the condition of *icterus neonatorum*. In its simplest form this probably results from the hæmolysis that reduces the number of red corpuscles so rapidly in the first week of extrauterine life, the only time of life in regard to which I feel justified in speaking of hepatic inadequacy in the absence of disease. It is a passing jaundice and rarely or never leaves any bad results. But occlusion of bile ducts and faults of development may also cause jaundice in the neo-natal period. Hepatitis with jaundice frequently occurs in babies from infection of the umbilicus, from intestinal infection or from a generalized septicæmic infection. If acute and severe, this infection results in acute necrosis or acute yellow atrophy, which may be rapidly fatal or may result in partial or temporary recovery. Many cases may be found in the literature in which apparent recovery has occurred, and autopsy a variable number of years later has disclosed some destruction of liver cells, with partial regeneration and subsequent cirrhosis. Many clinicians now believe that most cirrhoses of the liver in children are of this type and origin.

Weil's Disease.

A considerable increase in our knowledge came with the recognition of acute jaundice caused by infection by the *Leptospira icterohæmorrhagiæ*. Weil described the disease in 1886 sufficiently accurately to differentiate it from other conditions, although the causative organism was not isolated until some thirty years later. It occurs mainly among workers in damp, rat-infested premises, largely among waterside workers, sewer labourers, workers in the fish markets, and those on the canals in Holland; therefore it is uncommon in children. An acute systemic infection, it has a mortality of about 10%, but recovery is often incomplete with gradually progressive cirrhosis of the liver. It is probably more common in Australia than is generally supposed. As shown by serological investigation, the infection often occurs without jaundice.

Infective Jaundice not of Leptospiral Origin.

Hurst and Willcox, observing cases of jaundice in military camps, concluded that they were due to an ascending infection of the bile ducts by organisms of the colon group. Martin, however, at Lemnos, held that the infection, whatever the causative organism, was systemic and not due to direct extension from the bowel. In the camps the mortality reached 4% to 5%, generally from acute yellow atrophy, but it was found that the liver was often enlarged rather than atrophied and showed gross destruction of liver cells; and so the term necrosis came into general use. Time then showed that persons thought to have recovered sometimes progressed to increasing fibrosis of the liver and ultimately died of cirrhosis.

Catarrhal Jaundice.

An exactly similar process occurs in catarrhal jaundice in children. It is often supposed that catarrhal jaundice is a very minor children's complaint that requires little care or treatment. In Sweden it is a notifiable infectious disease and its mortality is 0·5% to 1%, but it has been noted that some epidemics are much more severe than others and carry a much higher mortality, chiefly from acute necrosis of the liver. Here again are found many cases in after years showing cirrhosis following the early necrosis of liver cells. In fact, catarrhal jaundice is simply the term applied to what seem to be the milder attacks of acute infective jaundice. In this country, too, we find some epidemics much more severe than others; that of 1932 proved particularly severe.

Toxic Necrosis.

Toxic necrosis is of a very similar nature, although here the action upon the liver cells is general and diffuse, rarely focal, and recovery seems possible only when the changes in liver cells do not proceed beyond the early stages of destruction. If recovery occurs at all, the end result is apt to be some fibrosis and a large, smooth, cirrhotic liver. It is suggested that the action of most poisons is effected via the hepatic artery, the large smooth liver being thus produced, while that of alcohol is one of chronic poisoning by way of the portal system, leading by its different incidence and varying degrees to a nodular cirrhosis.

Symptomatology.

In most cases the onset of the illness cannot at first be distinguished from the onset of other acute febrile illnesses or mild infections. After a few days of vague malaise, often with vomiting, abdominal pain and headache, jaundice appears, with yellow sclerotics, dark urine and pale stools. According to the severity of the infection this jaundice will disappear or persist continuously or with exacerbations for some months. With a complete disappearance of the signs and symptoms the case may be regarded as one of simple catarrhal jaundice, and forgotten. In many instances there may accompany the infection slight or considerable enlargement of the liver and often of the spleen and, even after the subsidence of these enlargements, there may occur vague malaise and sweats with or without slight recurrences of jaundice. A very large liver indicates a severe degree of necrosis of liver cells; if the enlargement subsides, the liver will exhibit an atrophy with nodular cirrhosis, or it may remain large, showing a smooth surface akin to the subacute hypertrophic biliary cirrhosis of Hanot. In any case if the liver has been large, it is unlikely that it can return to a normal state; some atrophy of liver cells and cirrhosis are inevitable. The later signs and symptoms, therefore, will be those associated with hepatic cirrhosis, gastric symptoms, hæmatemesis, ascites and dilated superficial abdominal veins. Cases giving the hæmolytic criteria usually accepted as those of hæmolytic jaundice may show at autopsy nothing but hepatic necrosis and cirrhosis.

Pathology.

The causation of the disease must be the entry of pathogenic organisms or of toxins or poisons to the circulation of the liver; and it is generally accepted that this may occur via the intestine as an ascending infection of the gall ducts, or by way of the lymphatics or abdominal veins, thence reaching the portal vessels and the liver cells; or it may be a direct infection of the arterial blood stream, reaching the hepatic cells by the branches of the hepatic artery. In Weil's disease the infection by the leptospira has been established as systemic; in cases of colon bacillus infection, or of infection by streptococci from a gangrenous appendix, or of infection of an umbilicus in a baby, it is a moot point how the organisms reach the liver cells. In cases of jaundice in babies and of catarrhal jaundice in older children who have died from accident during jaundice, it is known that there is definite hepatitis, which in the milder infections is believed to undergo complete cure. This must mean increased arterial blood supply, without which recovery is impossible. The normal sequence is increased blood supply through the normal hepatic artery and its branches; if this is not sufficient, there must be laid down new vascular fibrocellular tissue producing new vessels, new biliary canaliculi, and revitalizing liver cells or producing new cells. According to failure or success of this regeneration, will be the amount of fibrosis and the resulting avascular cirrhosis. The importance of a knowledge of the circulation of the blood within the liver becomes paramount if we are to understand the pathology. In the normal liver some two-thirds of the supply to the hepatic cells is provided by the portal vessels; in the diseased liver some two-thirds may be supplied by the hepatic artery. It is essential that the liver cells be supplied lavishly with arterial blood in order that revitalization and regeneration may occur. That the development of fibrous tissue is not merely to replace dead cells is shown by the fact that, in experimental occlusion of hepatic and portal veins at different times, although necrosis of liver cells occurs in each case, little or no fibrosis can be demonstrated.

I wish to emphasize the fact that the pathology is similar in all cases, inflammation or poisoning and necrosis of liver cells followed by attempts at regeneration, with increase of fibrous tissue in the organ. The site of incidence of the necrotic process, the amount of destruction of liver cells, the amount of regeneration, the degree of fibrosis and portal obstruction, and the degree of obstruction of the biliary passages, will all determine whether the liver will be large or small, smooth or nodular. To try to differentiate the portal from the biliary process is futile and misleading; the essential pathology is the same. The stage of development of vascular fibrocellular tissue with new blood vessels and new biliary canaliculi gives rise to the tawny yellow or reddish liver to which the term "cirrhosis" should etymologically apply. It is said that if one-eighth of the liver continues to function normally, the patient may remain in apparently good health.

Relationship between Liver and Spleen.—Much of the relationship between the liver and the spleen remains obscure. The enlargement of the spleen that occurs in most liver diseases brings this prominently before us, and Lord Dawson has stated that such splenic enlargement is the first clinical sign to be found in over 50% of cases of cirrhosis of the liver. While we know that jaundice is due to the liberation of bilirubin owing to the destruction of red blood corpuscles by the cells of the reticulo-endothelial tissue which is particularly active in the spleen, it is believed that one function of the liver cells is to alter the bilirubin in some unexplained chemical manner before it passes to the bile ducts. If the liver cells are diseased it passes into the systemic circulation unaltered; if there is obstruction to its passage to the duodenum the altered bilirubin is dammed back and again reaches the systemic circulation. This is the basis of the Van den Bergh reaction; its proof still awaits final investigation and confirmation.

Physiologically the spleen must be regarded as an organ of the vascular system; it otherwise shows very little evidence and no proof of pathological changes peculiar to itself. The fine fibrosis shown is probably merely a response to strain imposed by the dilated sinuses; it does not suggest inflammatory change. The reaction of the spleen to infections, its pallor in response to fright or fear, simply suggest that one of its chief functions is to regulate the supply of red blood cells and hæmoglobin to the general circulation. Any disease or disturbance of the reticulo-endothelial tissue, such as occurs in pernicious anæmia, leuchæmia, or the lipoid diseases, must disturb this and the liver shares in the general disturbance; it cannot, however, be said that any disease of the spleen alone has been proved to affect the liver.

In the middle of the last century cases were described exhibiting marked anæmia with splenomegaly; they were labelled "splenic anæmia". In 1882 Banti described a chronic type which he himself and some enthusiastic followers claimed as a new disease, although vague and variable pathological changes were reported. Banti held that even early cases showed cirrhosis of the liver and that the cirrhosis became more gross with the progress of the disease. Osler accepted Banti's description upon what seem insufficient grounds, but Rolleston, and later Hutchison, remained sceptical. These were inclined to regard splenic anæmia and the type designated Banti's disease as a mixed group, probably resulting from different causes; and they did not accept the fact of apparent cure by splenectomy as sufficient evidence of separate entity. Practically we find that cases diagnosed as splenic anæmia or Banti's disease ultimately prove to be of other nature, usually exhibiting on autopsy indications of damage of liver cells.

Blood Changes.—It is quite clear that no definite and pathognomonic blood changes can be expected in these cases. In acute cases, as would be

expected, there is generally a leucocytosis. Anæmia of secondary or microcytic type soon develops, and mononucleosis is common. Hæmolysis occurs very frequently, especially in the later stages, owing to an increased fragility of red cells, and many cases give the typical hæmological criteria of so-called hæmolytic or acholuric jaundice, under which heading most are grouped at some stage of their course.

Treatment.

The treatment of mild cases of infective jaundice, especially of the catarrhal type, with associated gastritis and duodenitis, necessitates complete rest in bed and administration of alkalis and glucose. I would emphasize the absolute necessity of rest and of watching the patient until he is quite well. So often the condition is diagnosed as "just jaundice", and some months later we find that the patient has never been well since. Flooding the liver with glucose seems to have some effect in aiding the recovery of liver cells.

Splenectomy.—Removal of the spleen was known in ancient times to have been resorted to in order to confer on athletes greater staying power and stamina. This is mentioned by Livy, but doubt has been thrown upon its value or even its stated purpose by recent investigations which show that its removal leads rather to more ready fatigue and lessened stamina, although the results in that direction are very slight. In any case it is heroic treatment for "stitch".

But many authentic splenectomies are known to historians. In Europe it was reported about 1549, but the earliest splenectomy in England was in Charles I's reign. Coming, however, to modern times, splenectomy was performed several times by Spencer Wells, but he unfortunately removed the spleen for leuchæmia with necessarily fatal results. It is only from 1908 onwards that splenectomy has been performed at all frequently, mostly for cases diagnosed as "splenic anæmia", "Banti's disease" and "acholuric jaundice". Most unfortunately and confusingly patients have been reported as cured within a few months of operation and only a very few have been traced over years. It is said that few are alive twenty years afterwards; yet at least one patient of Lord Dawson's was known, without operation, to be alive and reasonably well thirty-nine years after coming under treatment.

That the immediate effect of splenectomy in cases in which the liver is affected is dramatic is well known, but the results after two to ten years will depend entirely upon the amount of damage suffered by the liver cells. Probably the chief effect of splenectomy is to relieve the liver cells of the necessity of dealing with the products of splenic activity normally brought to them, and recovery of these cells may well be aided by early relief in this manner. In one case of my own splenectomy saved a boy dying of hepatic cirrhosis with ascites and general anasarca, for a period of eighteen months, during which he resumed school and afterwards factory life, ultimately dying, of course, from hæmatemesis. I believe that every patient should be given that chance and that splenectomy should be performed early. Prognosis should, however, always be guarded; the amount and progressive nature of the cirrhosis must always determine the after health and length of life of the patient.

Summary.

1. Attention is drawn to the occurrence, increasing frequency, and essential unity of the condition known as hepatic necrosis.

2. Doubt is thrown upon the occurrence of diseases of the spleen, commonly supposed to induce secondary cirrhosis of the liver.

3. Close study is urged of all cases of seemingly mild jaundice with a view to the discovery of possible ætiological causes.

4. Splenectomy is urged at an early stage of necrosis; the presence of definite cirrhosis of the liver should not be regarded as a contraindication to operation.

PRACTICAL METHODS OF MAINTAINING MUSCULAR TONE IN PREGNANCY.

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AT antenatal clinics and in private practice one cannot but be struck by the general flaccidity and loss of tone among women who have borne children. This is palpably wrong and is an instance in which prevention should be the ideal, rather than attempts at correction by later operative interference.

Having long held this idea, I express it with new confidence with the recent endorsement from the standard work of J. B. Mennell in "Physical Treatment by Movement, Manipulation and Massage" of 1934. He writes:

> It is certain that the amount of visceroptosis to be discovered amongst matrons in this country is a blot on the foresight of their medical attendants. There can be no excuse for the frequency with which a pathological change follows a purely physiological process. Nature could not be guilty of so grievous an error; therefore human management must be at fault.

If this is true—and it can hardly be disputed—the aim of the obstetrician must be not merely to prevent disasters and bring mother and child safely through the confinement, but, realizing that the process is a purely normal and physiological one, to return the mother to the community physiologically normal. The acceptance as inevitable of a certain degree of post-puerperal damage in figure and function, or of chronic pathological conditions, is obviously based on a compromise that is a confession of partial failure.

Mennell continues that the physical treatment of the obstetrical practice may be divided into three stages: during pregnancy, during labour, during the puerperium. The greater the experience in physical treatment, the greater the confirmation of its value in obstetrical work.

Since I wrote and offered this paper to the Queensland Branch last year, Margaret Morris, working for many years on similar lines in Saint Thomas's Hospital, has published her book, "Maternity and Post-Operative Exercises", 1936, with the full endorsement and support of Professor John Fraser, Professor R. W. Johnstone, Dr. Fairbairn and other leaders of medical thought upon this subject. I can heartily endorse from experience Dr. Fairbairn's[1] comment: "Physical exercises are an even more valuable restorative of muscular efficiency than massage, and still more neglected as a means of avoiding the abdominal wall and pelvic floor weakness that is so prominent a feature in the causation of inefficiency in women." The word "inefficiency" is physical, mental and functional in its implication.

One of the most obvious, yet one of the most subtle, differences between patients lies in that something which is called "tone". This is particularly noticeable among parous women. For example, a woman who has borne many children may still have that firm, elastic-feeling flesh, healthy-looking skin, and abundance of energy that go with good muscular tone. Another woman in but her second pregnancy may, on the other hand, show sagging tissues, lax abdominal wall and pelvic floor, varicose veins, swollen feet, sagging breasts, and an inelastic dryness of the skin texture and the general sense of fatigue that are indications of lack of tone.

Factors Affecting Muscular Tone.

What is this "tone", and what determines it in a muscular sense? Gould's medical dictionary calls tone a "normal degree of vigour and tension, a healthy state of the part". Sir Morton Smart[2] sums it up in the following way:

> When a muscle's elasticity, contractility and irritability are normal that muscle may be said to be in a state of tone, that is, it is in a state of attention to respond immediately to a physiological stimulus.

The tone of a muscle is determined by a number of factors: the nutritional quality and oxygenation of the blood that come to the part, the state and tone of its blood vessels themselves, the lymph drainage, and the innervation, the condition of the muscle fibres themselves and the quality of the cement substance between them. All these factors must be normal if good tone is to be maintained by a muscle—or by any tissue for that matter. Obesity is one manifestation of the failure of this tonal complex.

There is no doubt that the conditions of modern life, and especially of city life, tend to destroy the tone of the body, and of the abdominal musculature in particular. One of the first principles of physiology is that deterioration of function follows lack of use; and this most readily takes place in undernourished tissues. The sedentary occupations followed by so many young women and girls before marriage militate against good muscular development and tone.

The under-developed muscles of the abdomen and trunk are, in most women, pampered further by the almost universal use of corsets and other supports. Sport is perhaps a saving factor, but the proportion of girls engaged in sedentary or unstimulating occupations, entailing long standing without active exercise, who engage in regular sport, is small compared with the total number. In any case the exercise provided by sport for the wage-earning woman or girl is intermittent and inadequate to maintain good muscular tone. One of the most illuminating commentaries on muscular development is to watch a casual group of women run.

Over half a million women and girls, including girls from fourteen to sixteen, are engaged in industry, professions, trades or occupations in Australia.[3] Nearly 129,000 of these work in factories, 85·9% being in textile and clothing factories. There are thus many thousands of young women working in sedentary or monotonous occupations. A large proportion of those who are not wage-earners, though theoretically employed in domestic duties, do not get enough active exercise and little outdoor sport. The unbalanced type of exercise and well-recognized pelvic congestion produced by factory work, combined with the hurried and unbalanced diet of most factory workers, conduce to poor tone in those muscles most needed in child-bearing, and to an unhealthy condition of the pelvic organs.

The activity of the muscles is an important factor in determining the blood supply and the amount of nourishment brought to them. It does in fact play an important part in the efficiency of the whole circulatory system—arterial, venous and lymphatic. By their contractions and relaxations the muscles act as local pumping agencies for their own circulation as well as for that of neighbouring structures. It is estimated that the amount of blood that passes through a muscle when in action is at least six times greater than when at rest. When there is reduction of free blood supply to a part, waste products tend to accumulate in the tissue, and such accumulation further reduces efficiency of action.

Not only the quantity of blood and removal of waste products, but the quality of nourishment supplied to the cells has a definite effect. The diet of the mother therefore plays an important part in maintaining the tone of her tissues. It is well recognized that the diet of most Australians is overloaded with sugar and starch and is deficient in vitamins (probably in *B*, in *A* and in *C*), in certain minerals (probably lime, phosphorus and iron). Plimmer quotes the work of M. J. Rowlands, who found that with a relative deficiency of vitamin B_1, not sufficient to cause beri-beri, there was actual degeneration of the muscular coats of the bowel, with stasis and constipation and definite sagging of the stomach and colon below the normal position. Enteroptosis of this nature causes a constant dragging on the cœliac plexus. By acting as a dead weight such sagging abdominal contents would

further tire already relaxed abdominal muscles, so that they would afford little support to the abdominal contents. Both factors produce fatigue. If a sudden dragging on the cœliac plexus causes shock, a continuous drag on the sympathetic system over long periods would cause a constant drain on nervous energy, almost like a dripping tap, with easy fatigue and a sense of inadequacy, which the patient graphically describes as "feeling gone in the stomach". For the tone of the abdominal organs as well as for the supporting muscles, foods rich in vitamin *B* should take a prominent place in the diet of the expectant mother.

With regard to skin tone, McCarrison has shown that deficiency of vitamin *A* causes a dryness of the epithelium, and McCollum speaks of an early inelasticity and senility of the skin with vitamin-poor diets. Healthy skin shows a wider range of elasticity than does the dry, ill-nourished skin of those who live on an unbalanced diet. This is noticed particularly in the skin of the abdominal walls and thighs during pregnancy. If the patient is on a well-balanced diet and has firm elastic skin, quite severe stretching may take place in the later months without the production of those "striæ" which are so common and disfiguring a feature of pregnancy. Moreover, the skin remains firm and unwrinkled after the confinement has relieved the pressure.

It has been shown in England and Scotland that there is a high incidence of anæmia in pregnant women.[4][5] This is said to be a generalization applicable to city populations everywhere. All workers, both American and English, are agreed that, when untreated, anæmia is progressive as pregnancy advances and that it becomes more profound in successive pregnancies. On first principles, if part of the function of iron is to assist in the conveyance of oxygen to the tissues, a chronic anæmia must result in a continued condition of malnutrition of the muscular system, which is suddenly aggravated by pregnancy, parturition and lactation. The anæmia, being of the type due to a deficiency of iron in the diet, can be readily corrected by foods rich in iron, such as greens and liver, and by full doses of iron and ammonium citrate.

The diet of pregnant women is often severely restricted in regard to protein, particularly meat. Some systems of antenatal care state that meat should not be taken by the expectant mother except when advised by the doctor. If the woman should have any albumin in her urine or a slight rise of blood pressure, proteins are eliminated from her diet as far as possible, particularly in the later months. It is during these months that the demands of the fœtus for proteins are highest. May it not be that too drastic a reduction of these essential foods can cause an increased water retention[6] of the tissues, which, allied to "famine" or "war œdema", hampers the circulation and lymph drainage of the muscles, particularly in the most dependent parts—the legs, ankles and feet.

The minimal essentials laid down by the League of Nations Health Committee for the normal individual included a daily ration of seventy grammes of protein, and it was insisted that a "large proportion of this should be assimilable protein of animal origin". In pregnancy such provision is even more necessary, as the mother must create in the fœtus, placenta, fluids and membranes some 15% of her own body weight (taking 100 pounds as an average weight for an Australian mother). The protein ration should, therefore, not be cut down beyond the ordinary minimal limits if muscular tone and activity are to be maintained.

It has been demonstrated definitely that stokers' cramp, miners' cramp and similar disorders are due to a failure of tissue salts, particularly calcium and phosphorus and sodium. It has similarly been demonstrated that not only are the muscles thrown into spasm by this deficiency, but there are profound psychological sequelæ, varying from increased irritability and mental depression to frank hysteria.[7] The significance of an adequate proportion of mineral salts therefore, both for mental and physical health (if indeed these are not one and the same thing) needs no argument, particularly as these are manifestations which obstetricians will recognize as almost universal in some degree during pregnancy.

Recently much more attention has been paid to psychological factors in maternal welfare. During pregnancy the subconscious realization of a change in the centre of balance produces a sort of physical uncertainty that limits muscular movement. In many women there is actual unwillingness to do more than is essential in a muscular sense. This is expressed in a desire to lie down frequently. Under modern conditions this limitation may proceed to such a degree that the woman suffers from lack of exercise. She can overcome her sense of insecurity and gain her lost poise only by free use of her muscles and readaptation of the muscular sense to the new conditions by graduated exercises.

Professor R. W. Johnstone, in his preface to Margaret Morris's book, states that:

> In pregnancy and labour the mental outlook of the patient is of great importance, and here also these exercises are calculated to have a beneficial effect. The woman who deliberately exercises her mind and body along the lines of these simple bodily exercises is both directly and indirectly helping to secure for herself an easier and safer and less painful delivery.

Effects of Poor Muscular Tone.

Stretching of Muscles.—The *rectus abdominis*, external and internal oblique, and transversalis muscles become flaccid and stretch with pressure outwards and downwards of the abdominal contents. The result is not only a "loss of figure" and of general appearance—a condition which women dread—but far-reaching effects which favour visceroptosis and throw undue strain on the pelvic floor. On the tone and integrity of the pelvic floor depend the normal position and function of the pelvic organs. Flaccidity of the *levator ani* group of muscles has consequences only too well known

to gynæcologists—*prolapsus uteri*, retroversion of the uterus, cystocele, rectocele and their sequelæ.

Stretching of Fibrous Tissue and Fasciæ.—The white fibrous tissue which ensheaths the abdominal and perineal muscles and which itself forms an important part of the abdominal wall, is naturally to some extent dependent on the tone of the muscles to keep it taut. If the muscle is stretched, its fascia must be stretched also. The white fibrous tissue of which it is made contains no elastic or contractile elements and, once stretched, it can never regain its original shape. Moreover, the blood and nutrition of the neighbouring muscles must determine to a large extent the supply of the fascia also. As the fibres of the external oblique and transversalis muscles cease and the *rectus abdominis* becomes a narrow fibrous band some inches above the pubes, much of the weight must be borne by the fascial aponeuroses of the lower part of the abdomen.

The posterior layer of the rectus fascia, however, ceases abruptly at the semicircular line (at the junction of the middle and lower thirds of the muscle). From there down the muscle is in direct contact with the transversalis fascia, which in this situation is frequently very thin. Thus the fascial supports of the abdominal wall are at their weakest at those very points on which the weight of the heavy abdominal contents must fall increasingly. It is not surprising, therefore, that the fasciæ of the lower third of the abdomen tend to stretch with repeated pregnancies and that support in this region is beneficial when the muscular tone is poor, or when the fœtus is large, or when heavy work has to be undertaken.

Stretching of the Skin with the Formation of Striæ Gravidarum.—In many women, as well as showing striæ, the skin becomes so flaccid and wrinkled that only partial restoration of its tone takes place. Some women go through many pregnancies without developing these disfiguring marks; and I believe they can be lessened or prevented altogether by maintaining a good nutritional tone of the muscles and a good blood supply to the skin, by exercise and massage of the abdominal wall with oil.

Varicose Veins.—The blood supply of the pelvis and lower extremities is greatly increased in pregnancy and the vessels are fuller than at other times. Pressure by the pelvic contents on the engorged iliac veins at the brim of the pelvis, combined with a lax abdominal wall and pelvic floor, impedes the return of blood from the lower extremities and from the vulva, with the consequent formation of varices. Poor tone in the leg and thigh muscles affords little support to the walls of the veins and lymphatics, so that as they are already overfull, further varicosity of the veins is favoured, with swelling of the feet from the impeded lymph return. Professor Lee tells me that stasis of the circulation in the lower half of the body is one of the main causes of the extreme fatigue felt by these women.

Means of Maintaining Muscular Tone.

A Well-Balanced Diet Rich in Minerals and Vitamins.—The diet advocated in the leaflet published by the Nutrition Subcommittee of the Queensland Branch of the British Medical Association supplies all needs. When, however, a deficiency is suspected to have been present in the diet, those elements which were lacking should be taken more freely in the daily food; or when the deficiency is marked it can be made good by some of the concentrated elements, of which there are so many on the market.

Fresh Air and Sunlight.—The medical attendant does not always realize that many women spend most of their days in the house or on verandas, and consider that being on the veranda is equivalent to being in the open air. As pregnancy advances women will venture to take their daily walk only in the evening. Consequently they seldom come in contact with direct sunlight—so important a factor in maintaining bodily tone. Expectant mothers should be encouraged to spend as much time out of doors as possible. Sensitiveness on the score of going out in the day-time is an attitude of mind that shows an entirely wrong scale of values and should be corrected by an understanding medical adviser. The expectant mother should go about naturally and happily among her fellows, following her normal avocations and enjoying life. To remain cloistered indoors during the later months of pregnancy not only promotes physical depression, but also mental depression and stagnation that are perhaps the worst enemies of good muscular tone. Some part of each day should be spent in the sun, and exercise should be taken in the open air when possible.

Exercise.—The value of ante-natal exercise is no new idea. It was, indeed, advocated by Aristotle about 2,400 years ago ("Book of the Generation of Animals", IV, 6). Even at the present time the idea is widespread not only in civilized countries, but among peoples who are more primitive, and special exercises are carried out by pregnant women in places as far apart as Persia, Patagonia and the Andaman Islands. In modern times in England, before the War, Dr. Fairbairn suggested ante-natal exercises as well as massage, though post-natal exercises only were more in favour at that time.

Exercise is the best stimulant of muscular tone. The blood supply is increased, more food and oxygen are brought to the muscle and its nerves, and fatigue products are carried away. Exercise in pregnancy should stimulate the whole muscular system and in addition be directed to special muscles on which the strain of both pregnancy and confinement will fall. It should never be of a type that might damage the fœtus or favour miscarriage. It should never fatigue, but rather stimulate, and should be graduated so that the muscles become educated in suppleness and endurance.

As every woman has a different capacity for exercise and a different response, her own feelings should

be her guide to some extent. Anticipatory fatigue, however, should be no bar to the institution of the exercises. Many types of sport to which the woman is accustomed might well be continued during the early months, unless, or until, she feels some discomfort in playing them.

Cramp in the legs, particularly in the calf muscles, is often a very severe trial to a pregnant mother, and is a not infrequent cause of disturbed sleep. Five minutes' general massage applied to both legs, percussion of all sorts being prohibited, will probably do more than anything else to alleviate this distressing symptom, and usually it will be found to relieve it altogether.

Support.—The use of a maternity corset or support is an admission of partial failure in maintaining muscular tone. But a support should always be advised whenever the patient feels the need for it. Muscular and general tissue tone should be sufficient to keep the abdominal and pelvic contents from bearing too heavily on the walls of the cavity, but contractile muscular tissue is not the only factor to be taken into consideration.

After the sixth or seventh month, many women, especially *multiparæ*, are greatly comforted, their fatigue is lessened and their general sense of well-being and efficiency are fostered by wearing a suitable abdominal belt or corset. There are numerous corsets on the market, but nearly all are too rigid with bones, and tend to compress instead of only supporting from below upwards. The weight should be suspended from the shoulders, not from the sacrum and pelvic girdle, and shoulder straps should always be attached to the support after the seventh month. Many women cannot afford the expense of even moderately priced maternity corsets; the abdominal belt made of strong calico, illustrated and described in the appendix to this paper, is quite sufficient and very cheap; the cost, with buckles and suspenders, is two shillings and sixpence. It has proved useful enough to be adopted by the Department of Maternal and Baby Welfare in New South Wales and Queensland, and incorporated in the Queensland "Mothers' Book" and the New South Wales booklet "Healthy Motherhood".

Post-Natal Care.

Post-natal care of the muscles is quite as important a factor as ante-natal care in returning the mother to normal life. Mennell,[10] Margaret Morris[8] and Van de Velde[9] are most insistent on the value of graduated exercises as well as massage, and have worked out systems of treatment for the puerperium and after. Following a scathing indictment of usual post-natal treatment, Mennell writes:

> The abdominal wall has been severely stretched; then the stretched muscles are placed under the most unwonted strain; it may be for hours. Small wonder, then, that when the stretching is relieved and the strain is over, the muscles simply "give out" and remain flabby and torpid. The muscles are naturally "stiff" and sore after their unwonted exertion, and the earliest attempts to contract them will cause some pain or discomfort. This will act as an inhibitory agent and, unless something is done to counteract it, the patient will leave them in a state of flaccid inhibition from which, not uncommonly, they never recover.

For the majority of patients a system of mild exercises in the recumbent position, such as those in "Schedule C", published in the appendix to this paper, can be performed cautiously after the first week in normal cases, though easy foot and ankle exercises can be carried out from the second day. These, combined with the frequently adopted postural treatment, such as the adoption of Rollier's position, sleeping in the ventral position, adoption of the knee-chest position, the special exercise for contracting the *levator ani*, will promote involution and maintain muscular tone of both the abdomen and pelvis during the period of enforced rest in bed; while physical culture at home or in classes might well be continued for several months, till the figure has regained its normal lines.

Massage and exercise conducted by a skilled masseuse are valuable aids to involution and to a rapid regaining of strength and figure after the confinement. For all those who can afford the extra expense it is well worth while.

Summary.

Owing to the conditions of civilized life, its lack of muscular activity, indoor life, sedentary occupations, unbalanced diet and almost universal use of corsets and an artificial outlook on pregnancy, the muscular tone of modern women is not good; particularly is it poor in those muscular systems which bear the brunt of pregnancy and labour. The consequences are not only unæsthetic, in loss of figure and good looks, but loss of muscular tone and stretching of fasciæ are responsible for a large proportion of the gynæcological troubles which hamper the woman's efficiency in her married life, in regard to future pregnancies, and in her normal everyday life; they sooner or later demand operative interference.

Most of these disabilities are preventible. If the causes of the loss of muscular tone are combated by a good diet, by an active life with outdoor exercise and sunlight, by ante-natal exercises specially designed to maintain the tone and control of the abdominal and pelvic muscles, by judicious support when necessary in the later months of pregnancy, and by post-natal exercises, by postural treatment, and, if possible, by massage, then the natural physiological processes of pregnancy and childbirth should leave the health, poise and efficiency of the woman not only unimpaired, but definitely benefited by the experience.

References.

(1) Fairbairn: "Changes in Thought in Halfcentury of Obstetrics."
(8) Morton Smart: "Manipulative Surgery", "Medical Annual", 1936.
(9) Commonwealth Year Book, 1934.

(46) L. S. P. Davidson, H. W. Fullerton, and R. M. Campbell: "Nutritional Iron-deficiency Anæmia, with Special Reference to Prevalence and Age and Sex Incidence", *British Medical Journal*, August 3, 1935, page 195.

(47) M. Davis and E. W. Walker: "Anemias of Pregnancy", *New England Journal of Medicine*, Volume CCX, June 21, 1934, page 1315.

(48) Kohman, Epstem and Peters: Quoted by Louis Berman in "Food and Character", 1933.

(49) Margaret Morris and M. Randall: "Maternity and Post-Operative Exercises", 1936.

(50) *Ibidem.*

(51) Van de Velde: "Sex Efficiency Through Exercises."

(52) James B. Mennell: "Physical Treatment by Movement, Manipulation and Massage", 1934.

Appendix.

Directions for Making a Maternity Belt.

Take a strip of strong unbleached calico about one and a half yards long and ten to twelve inches in width; thinner material can be used double. Pin calico round the lower part of the abdomen and fit it to the figure with darts at the upper and lower edges (see diagram), leaving a good lap-over for expansion. Do this lying on the bed in a recumbent position. When you stand, the belt will give the support you need. Now hem or bind the edges. On to the wrapped over end sew four or five strips of webbing or calico bands, and on to the tucked under side sew a corresponding number of buckles (the kind used on the sides of men's tennis trousers—very cheap). Put the buckles in such a position that when done up the belt gives comfortable firm support, not tight pressure. The belt should be worn low in front, and have suspenders attached to its lower edges to keep it down and the stockings up.

This support is sufficient for the fifth and even sixth month, but as soon as the need is felt, shoulder straps should be added for further support from the shoulders. These can be made of wide bands of webbing or folded calico. If too narrow, they cut the shoulders. Sew on the straps each side of the mid-line in front several inches down on to the belt to prevent tearing; they cross in front, go up over the shoulders, cross at the back, and are stitched on to the belt at the back about two inches each side of the mid-line. Such a support costs but a couple of shillings, but gives great support and comfort during the later months of pregnancy. It is most necessary in the morning when doing the housework, and should be loosened for the afternoon rest and adjusted again before rising. Varicose veins, cramps, swollen feet, backache, heaviness, dragging pains and tiredness are all relieved by the wearing of such a support.

Schedule C: Exercises in a Recumbent Position.

These are specially adapted for the later months of pregnancy and for delicate mothers.

1c: Lying flat on the back without a pillow, raise the arms slowly sideways above the head, taking a deep breath and filling the lungs. Hold the position for a couple of seconds and lower the arms. Repeat four times. Rest.

2c: With the knees slightly bent and the heels resting on the bed raise the head, but not the shoulders, from the bed as if trying to look at the toes. Nod the head once and lower to the bed. Do this ten times to start with, increasing to twenty. If the hands are placed on the abdomen, the contraction of the muscles will be felt with each rise of the head. This exercise is particularly strengthening to the abdominal wall.

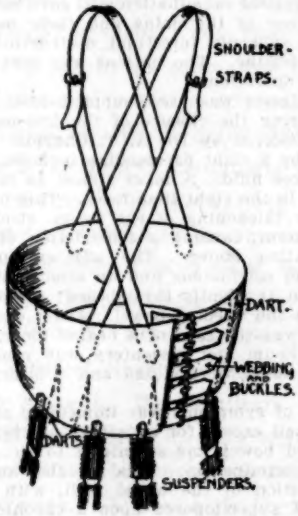
3c: Still lying flat on the bed, raise the right shoulder and try to touch the left knee with the right hand. Repeat with the left hand towards the right knee. Repeat two to four times.

4c: Keeping the shoulders and heels on the bed, raise the right hip, twisting the body to the left. Now raise the left hip, twisting the body to the right. Repeat each two to four times.

5c: Lying on the back with a low pillow under the head, bend one knee and bring it up towards the chin. Take hold of the side of the heel with the hands and straighten out the instep. Now stretch the leg out with some vigour, drawing the palms of the hands firmly up the back and sides of the legs, continuing the rubbing movement up over the abdomen and breasts. Slide the hands again down to the ankle as the knee is raised again. Repeat two to four times with each leg. This exercise is specially helpful in cases of varicose veins, swollen feet and cramps.

6c: Draw in the stomach wall and let it relax—about twenty times quickly. The easiest way to learn this exercise is to contract as you breathe out and relax as you breathe in. But as control of the muscles is improved the movements can become independent of the breathing. It can be performed lying on the back, then lying on each side, and finally standing.

7c. Lying on the back with the feet about eighteen inches apart, stiffen and contract the muscles of the buttocks, stomach wall and inner sides of the thighs so that the whole body is rigid. Relax completely, stiffen, relax. Repeat four times, increasing to eight. Vary by performing exercise lying on the back, first with knees straight and then, as proficiency is gained, with the knees bent.



8c: Exercise for mobility. Lying on the back, bend one knee, keeping the foot on the bed. Let the knee fall loosely sideways on to the bed and straighten. Repeat four times with each leg.

9c: Special exercise for pelvic muscles. Take in a deep breath, now breathe out, forcing all the air out of the lungs with a steady contraction of the muscles of the stomach and pelvis until you feel as "tucked up" as possible and the buttocks are contracted too. Then let the muscles relax and breathe naturally again. Repeat several times morning and evening. With practice enough control is gained to contract the muscles without having to expel the breath each time.

There should be a short period of relaxation after each exercise and a longer one after the breathing exercises. Each set of exercises should take about ten to fifteen minutes, and at the completion there should be a sense of invigoration, but no real fatigue. The muscles will probably be stiff at the beginning, but if the exercises are persisted in the stiffness will pass off in a few days. A rest for five to ten minutes should always be taken lying on the bed at the completion of the exercises.

Reports of Cases.

REGIONAL ILEITIS.

By VICTOR J. KINSELLA,
Sydney.

REGIONAL ileitis is not a common condition; but a knowledge of it may, on occasions, prove of great importance to the general practitioner, surgeon and radiologist. My attention was drawn to it through the following case.

Report of a Case.

On January 2, 1932, I was called in to see Mrs. E.K., aged sixty-five years, who had been suffering for some hours from abdominal pain and vomiting. The pain was central, severe and colicky. The bowels had been well opened the day before. Two weeks before she had suffered from diarrhœa. For many years she had had similar attacks of colicky abdominal pain and vomiting ("gastritis"), but none so severe as the present. Examination revealed no abnormality beyond some indefinite abdominal tenderness. An enema was ordered, and this was followed by a satisfactory evacuation. But the pains and vomiting persisted. Next day another enema was given. Only a little fæcal stained fluid, with no flatus, returned. Physical examination still gave indefinite results. The persistence of the pains and their nature led to a diagnosis of subacute intestinal obstruction, probably in the small intestine. The patient was sent into Dalcross Hospital for operation.

Spinal analgesia was used, supplemented, on account of straining during the closure of the abdomen, by a little ether, administered by Dr. A. T. Chapple. The abdomen was opened by a right paramedian incision. There was a little clear free fluid. A mass almost as large as a hen's egg was felt in the right iliac fossa. This proved to be an inflammatory thickening of the ileum, about seven centimetres in extent, causing a constriction of the gut with slight dilatation above. The adjacent mesentery was thickened and œdematous and an almost black vein with no circulation (evidently thrombosed) could be seen in it, passing from the affected bowel. About eight centimetres lower down was the scar of a healed ulcer. The affected segment of ileum and mesentery was removed, the two ends of the ileum were closed and a lateral anastomosis was made.

The relief of symptoms was immediate and the patient progressed well except for an attack of cystitis.

The excised bowel was submitted to Dr. Leslie Utz for microscopic examination. This revealed an acute inflammatory condition of the bowel wall, with small hæmorrhagic ulcers, superimposed upon a chronic inflammatory or fibrotic condition, which did not suggest tuberculosis.

Eight months later, the first account of regional ileitis was published by Crohn, Ginsburg and Oppenheimer. They clearly differentiated the condition from other chronic inflammatory changes occurring in the ileum. Since their paper, numerous accounts of cases have appeared, especially in the American literature.

Pathology.

The cause of the condition is unknown. The terminal part of the ileum is found at operation to be thickened and œdematous, the change often extending to the ileocæcal valve. As a rule only the terminal 25 to 35 centimetres (10 to 14 inches) are involved; but any part of the small or large bowel may be affected. The serosa is blotchy-red in colour, and oval mucosal ulcerations are found in the mesenteric border, lying in the long axis of the intestine. Microscopically, various grades of acute, subacute and chronic inflammation, with no specific features, are found. The submucosa and, to a much less extent, the muscular layers, are the seat of marked inflammatory and exudative changes. Later, fibrosis and stenosis occur. There is a marked tendency to chronic perforation, with abscess and fistula formation.

In the past the condition has been confused with ileocæcal tuberculosis. But, as a rule, there are no giant cells, and no test reveals tubercle bacilli.

In October, 1936, Reichet and Mathes published an account of interesting experimental work in which they had produced similar lesions in dogs by injecting into the lymphatics of the intestine various sclerosing agents, with and without bacteria. They suggest that the causative factors are low-grade infection together with a chronic lymphœdema due to lymphatic obstruction.

The Clinical Appearances.

Young adults are mostly affected. Many of the patients (half of them in Crohn's series) had been subjected to appendicectomy. Clinically the cases fall into four groups:

I. Cases indistinguishable from acute or subacute appendicitis.

II. Cases with symptoms of ulcerative enteritis—abdominal pain, diarrhœa, with mucus, blood and pus in the stools, and loss of weight and pyrexia. Sigmoidoscopy and X ray examination after a barium enema exclude colitis. After a barium meal X ray examination shows ileal changes.

III. Cases with symptoms of subacute or acute intestinal obstruction. These may be the first symptoms of the disease, or there may have been previous inflammatory symptoms such as are indicated under groups I and II.

IV. Cases with fistulæ, sometimes following the drainage of an abscess, thought to be appendiceal. But the appendix is found uninvolved.

Radiographic Appearances.

Radiologically, the condition may be overlooked unless the clinician suggests that it be specially sought for. It requires repeated screening and skiagraphy, three, six and nine hours after the ingestion of the meal. In the early stages of the disease there is delay in emptying the terminal coils of the ileum. Later there is definite dilatation of the subterminal coils, and from these dilated coils a thin irregular stream of barium may be found passing through a filling defect to the cæcum. This "string sign" is characteristic. A palpable mass may be found corresponding with the filling defect.

Treatment.

Resection of the affected part is the treatment of choice. If this cannot be done it may be necessary to carry out ileo-colostomy, with exclusion, by dividing the ileum proximal to the diseased portion, closing both ends, and implanting the proximal end into the colon beyond the diseased portion. Later it may be possible to carry out resection as a second stage.

Reviews.

FUNDAMENTALS OF BIOLOGICAL CHEMISTRY.

THE fifth edition of Parsons's "Fundamentals of Biochemistry" is sixteen pages longer than the fourth edition, but the changes in the text are not numerous.[1] The greater part of the book is essentially unchanged, a sentence here and there having been added for greater clearness or correction. The section on the chemistry of muscle metabolism has been considerably altered to bring it into line with recent work. The flavines, the constitution of the sex hormones and the constitution of some of the vitamins are new subjects treated.

The book is the best introduction to biological chemistry available in the English language. It is surprisingly complete and is written in a very entertaining style. A useful bibliography is given at the end of each chapter to enable further reading to be done. It can be highly recommended to anyone who wishes to know something of the present position of biological chemistry but who is not inclined to read the larger text-books.

[1] "Fundamentals of Biochemistry in Relation to Human Physiology", by T. R. Parsons, B.Sc., M.A.; Fifth Edition; 1935. Cambridge: W. Heffer and Sons, Limited. Crown 8vo, pp. 465. Price: 10s. 6d. net.

The Medical Journal of Australia

SATURDAY, MAY 29, 1937.

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MALIGNANT DISEASE OF THE COLON.

ONE of the most useful methods of learning the state of knowledge of any subject in medicine is to review the literature over a period of one or more years. The procedure is laborious and time-consuming, but the results are generally worth while. It might be thought that little that was new could be learned on such a subject as malignant disease of the colon, for the pathology of the lesions is clear, the symptoms are set out in any up-to-date text-book, the methods of examination are those of everyday practice, and most experts are agreed as to the value of different forms of treatment. But even the most experienced, be they physicians who diagnose malignant conditions, or surgeons expert in their treatment, can learn something from the experience of others. The general practitioner also, who but rarely and unexpectedly sees a malignant bowel condition, will find that the collective experience of many writers is an incentive to watchfulness as well as a reminder of the relative value of certain signs, symptoms and forms of treatment.

C. F. Dixon, of the Mayo Clinic, has made a review of the literature on malignant disease of the colon from July, 1935, to July, 1936.[1] He has dealt with about one hundred articles from English, Spanish, French and German journals, and some of the points that he makes are of importance to the clinician. Many observations have been made on the relationship of polypi to malignant disease, and it is held that rectal adenomatous polypi constitute the most dangerous as well as the most common predisposing factors in the development of rectal adeno-carcinoma. One observer found that malignant changes occur in more than 40% of polypi; another found that 70% of the malignant lesions in 130 specimens removed at operation from the large intestine had an undoubted relationship to intestinal polypi; another attributed the development of carcinoma of the large intestine to polypi in as high as 63% of cases; and so on. Divergent opinions are held in regard to the part played by heredity in human carcinoma; and most observers will agree with Dixon in his contention that there is justification for the tentative acceptance of an inherited predisposition, though many will go much further. There is uniformity of opinion about the occurrence of carcinoma in the various segments of the bowel. About a third of the tumours are in the right half of the colon, a third are in the sigmoid and a third are in the transverse segment and the left side of the colon. From the point of view of diagnosis greatest interest centres in the symptoms and signs of the condition. Steindl found that in 22% of a series of cases of inoperable carcinoma the condition became inoperable because of errors in diagnosis. Unfortunately the beginning of the disease and the occurrence of the first symptoms seldom coincide; and even when carcinoma of the colon does cause a disturbance, the symptoms are not always sufficiently definite to allow a diagnosis to be made. Dixon finds that when carcinoma is present in the caecum or in the right side of the colon, the commonest error in diagnosis is unexplained secondary anæmia, pernicious anæmia, peptic ulcer, cholecystic disease or appendiceal abscess. If the growth is in the left side of the colon the false diagnosis is likely to be appendicitis, colitis, spastic colon or disease of the uterine adnexa. Colitis and hæmorrhoids are most likely to be the wrong diagnosis when the growth is

[1] *Surgery, Gynecology and Obstetrics*, December, 1936.

in the rectum. Cade holds that rectal examination and the intelligent use of X rays should permit early diagnosis in 90% of cases. He also holds—and not nearly enough stress is laid on this—that the reason why only 40% of the lesions are recognised early is that the medical attendant is unable to convince many patients of the necessity for a thorough examination. It is also occasionally forgotten that some growths become inoperable because patients refuse to submit to laparotomy when a diagnosis has been made.

The questions of special diagnostic and operative procedures need not be discussed; they are not called for until the presence of malignant disease has been suspected by the regular attendant. If the medical attendant has made an early diagnosis the patient will be in a sufficiently good condition to undergo the ordeal of treatment. The responsibility of the medical attendant does not end with the making of the diagnosis; he has to see that the patient receives the best treatment that is available. The greatest skill and most mature judgement are required for the successful treatment of malignant disease of the colon; and most general practitioners know where these qualities are to be found.

Current Comment.

COLLAPSE THERAPY IN PULMONARY TUBERCULOSIS.

ATTENTION has often been drawn in this journal to the value of artificial pneumothorax in the treatment of pulmonary tuberculosis. It is widely recognized that this specialized method, whereby the lung is made to collapse, or, as modern writers prefer to put it, is relaxed, is of the greatest value, in spite of the fact that it is not free from troublesome and even serious complications, and is an arduous treatment for both doctor and patient. The question at issue in recent times is rather what are the indications for it—whether it is a method suitable only for lesions of slight extent in patients relatively free from toxic manifestations, or whether it is to be used whenever collapse of the lung may be safely induced and there is no risky degree of contralateral disease. G. L. Leslie and R. S. Anderson answer this query in no uncertain fashion when they record a series of 1,124 patients in one large sanatorium, no fewer than 78·8% of whom were actually submitted to treatment by artificial pneumothorax.[1] Of these patients, 823 were discharged from the sanatorium; the others were still in residence at the time of writing. Of the former, arrest or apparent arrest of the tuberculosis was obtained in 57%, and either decrease in size or obliteration of cavities in 69%, while tubercle bacilli were no longer found in the sputum of 59% of those patients in whose sputum they had previously been present. In 70% of the discharged patients tubercle bacilli cannot now be demonstrated, a fact of great importance in prophylaxis. But of those patients treated by pneumothorax, no fewer than 80% had "negative" sputum after discharge. This article of Leslie and Anderson is highly statistical, but even if we remember the dictum of Burrell, that the results of a specialized form of treatment in so variable a disease as pulmonary tuberculosis cannot well be submitted to the test of figures, we cannot help being favourably impressed. Let us assume that we do not know what type of case these physicians treated, or even that numbers of them were of a very favourable kind, can the figures be bettered by other methods? Leslie and Anderson claim that their results are much superior to those obtained in other institutions where relatively little attention is paid to collapse therapy. Further, they state that their studies confirm them in a belief that there is an "overwhelming argument in favour of a definite policy of early and intensive collapse therapy for approximately three-fourths of the patients with the adult type of pulmonary tuberculosis". More conservative opinion may not accept this statement in its entirety, but in conclusion let us ask three questions: Can any physician be sure that the earliest and slightest of pulmonary lesions may not flare up into a dangerous activity even under good conditions? Is the decision to induce artificial pneumothorax made in a sufficient number of cases at a favourable stage when it is easily possible? Lastly, are the sanatoria of this country adequately equipped to carry out this important adjunct to the treatment of pulmonary tuberculosis?

MÉNIÈRE'S DISEASE.

SINCE Ménière in 1861 first described the syndrome that bears his name, its complete pathology has remained obscure, in spite of many explanations. To the patient a seizure is an extremely terrifying experience and is attended by a fear of impending dissolution. Even to the physician death may appear imminent. Medicinal treatment is futile. The drugs employed have been legion in number, both pharmacopœial and extra-pharmacopœial, but they are in the main entirely useless. Surgical intervention has claimed a proportion of successes, although fraught with considerable hazard. The sufferer's plight, however, may be so pitiful that he would resort to any measures that would give a reasonable prospect of

[1] *The American Journal of the Medical Sciences*, February, 1937.

amelioration. The condition is not usually followed by death, and consequently *post mortem* findings are uncommon. It has been included amongst the paroxysmal neuroses, but definite pathological lesions have been found. These, however, have varied in the different cases, the essential factor being pressure upon the auditory nerve. In view of the reference to Ménière's syndrome in Dr. Phillips's paper appearing in this issue, it will be useful to refer to a recent article on the subject by W. E. Dandy.[1]

In Ménière's disease and pseudo-Ménière's disease Dandy is an enthusiastic advocate of section of the auditory nerve or its vestibular branch alone. His series of operations now reaches 170 in 160 patients, ten having been bilateral. He states that there has been no death. His first patient, however, died after the left vertebral artery was ligated and the right vertebral artery was compressed. Dandy asserts that Ménière's original description of "a serosanguineous exudate in the semicircular canals" was not from a case of Ménière's disease at all, but from one of acute suppurative labyrinthitis. In the present communication Dandy describes eight patients, all satisfactorily operated upon, except the first, as already indicated. Dandy's cases demonstrate the production of Ménière's syndrome by tumours and aneurysm of the basilar artery. He also gives a series of five large arterial loops (from the antero-inferior cerebellar artery) in the lateral cistern, which he considers to be equally positive lesions producing the syndrome. These Dandy believes to act by compressing or strangling the auditory nerve. He states also that among those coming to operation in the past year, about 10% showed contacts from large arteries and that probably in addition there are smaller vessels producing the same effect. Characteristic Ménière attacks have been associated with tumours in the cerebello-pontine angle. However, Frankl-Hochwart (1898) was not convinced that the effect of the tumour on the eighth nerve was responsible for the attacks, because injury to contiguous areas of the brain was too widespread. He further considered it doubtful whether isolated disease of the auditory nerve could give rise to Ménière attacks, as no known instance had been reported. Dandy's impression had always been that dizzy attacks with acoustic tumours were rare, but of seventy-nine cases in which that particular feature was mentioned, nearly 80% of patients actually gave a history of dizziness. Dandy is unable to say what proportion of these had real, recurring, sudden attacks of dizziness like those of Ménière's disease. The reason why the erroneous belief is held that dizzy attacks are uncommon with acoustic tumours is explicable from the fact that dizziness is only a minor part of a group of severe manifestations which are usually sufficient to establish a diagnosis. Also dizziness may disappear when the nerve function is lost owing to the growth of the tumour. In Dandy's whole series of cases of Ménière's disease an actual tumour was never disclosed unless there were signs and symptoms indicating the presence of a tumour irrespective of dizziness. It must not be forgotten that minute tumours may exist in the auditory nerve which are not disclosed at operation or which are missed in gross inspections at autopsy. Dandy rightly calls attention to the fact that gross lesions may exist in the auditory nerve and still escape detection.

Dandy has called attention to evidence that arterial contacts with the bare sensory root of the trigeminal nerve in the posterior cranial fossa are responsible for most cases of trigeminal neuralgia. He thought that similar lesions might explain Ménière's disease, but it is more difficult to arrive at such a conclusion, because there may be no gross vascular abnormality. The eight cases presented in this treatise, however, require no argument. Only five other vascular lesions on the auditory nerve have been selected. In two of these arterial loops have apparently strangled the auditory nerves, and in three a large looping artery, free in the *cisterna lateralis*, has lain upon the nerve. The five large arteries reported in this communication, with the exception of one, represent the positive results gained from last year's experience only. Dandy considers that the number of large compressing arteries is approximately 10%. Probably in from 8% to 10% of cases of Ménière's disease symptoms are bilateral, and these could not be explained by bilateral lesions along the auditory nerves. The only explanation of bilateral cases must be an intrinsic lesion of the auditory vestibular pathways in the brain stem. As far as other lesions reported in the literature on Ménière's disease are concerned, K. Wittmaack found concretions in the *ductus cochlearis* of two patients, in one of whom there was also a minute neuroma in the cochlea. In another case a small hæmatoma was found in the brain stem near one of the nuclei of the auditory nerve.

Dandy considers that attacks of Ménière's disease are analogous to those of epilepsy and trigeminal neuralgia. He holds that only lesions of the sensory root of the auditory nerve (its vestibular division) can cause Ménière's disease. Therefore, lesions in the semicircular canals, such as concretions, would not be causal factors. Again it is difficult to understand how occlusion in the *ductus cochlearis* could affect the semicircular canals. Of the theory of G. Portman and H. Aboulker, that pressure effects are transferred from the *cisterna lateralis* through the *ductus endolymphaticus*, Dandy has no doubt. He insists that pressure in the lateral cistern does not affect the semicircular canals or organ of Corti, as is demonstrated by the absence of effect from brain tumours that are known to cause rise of pressure in the lateral cistern without any pathological effect on hearing or vestibular function.

Whether heroic measures are justified for Ménière's disease is a moot point. Furstenberg's treatment, described by Dr. Phillips, is simpler, but whether it would be effective in the presence of gross pathological lesions is doubtful.

[1] *The Journal of the American Medical Association*, March 20, 1937.

Abstracts from Current Medical Literature.

GYNÆCOLOGY.

The Treatment of Functional Uterine Hæmorrhage by Gonadotropic and Ovarian Hormones.

T. N. A. JEFFCOATE (*The Journal of Obstetrics and Gynæcology of the British Empire*, February, 1937) has carried out an inquiry into the value of gonadotropic and ovarian hormones in the treatment of functional uterine hæmorrhage. *Questionnaires* were sent to leading gynæcologists in every country in the world and their experiences are embodied in the paper, together with the observations of the author and his colleagues of the department of obstetrics and gynæcology of the University of Liverpool. The paper is an exhaustive study of the present position in regard to the use of ovarian and gonadotropic hormones in uterine hæmorrhage. The author discusses all the various causes of uterine bleeding, both normal and abnormal, and the basis of treatment. The results of treatment are tabulated. Only 34 surgeons from the leading clinics of the world use organotherapy in this connexion. The general opinion is that the use of ovarian and gonadotropic hormones in functional uterine hæmorrhage does not give the brilliant results frequently claimed for it. It is only in conditions associated with endometrial atrophy or menopausal menstrual irregularity or slight loss at the time of ovulation that œstrin will produce luteinization. The preparations of *corpus luteum* would seem to give as good results as the gonadotropic hormones. *Metropathia hæmorrhagica*, the non-ovular type of hæmorrhage, gives the best response to organic preparation. Hæmorrhage at and about the period of puberty responds very well, but hæmorrhage of the menopause does not give satisfactory results. Of hæmorrhages in puberty, 80% are controlled by ovarian extracts. Treatment in most cases gives only a temporary result.

Genital Prolapse.

A. MAYER (*Monatsschrift für Geburtshülfe und Gynäkologie*, October, 1936) discusses various details in technique which should be observed in operations for uterine prolapse. The first concerns tightening of the bladder sphincter in the repair of a cystocele. Urinary incontinence is so frequent that the tissues round the urethra should be firmly united and so help in restoring vesical control. Next comes the best method of dealing with the cystocele. The bladder should be pushed up until the vesico-uterine fold of peritoneum is exposed, and laterally until the fascial layers can be clearly determined. The author considers that many operators do not actually expose the *levatores ani* during posterior colporrhaphy, and he insists on the importance of this matter. If these points be observed, the uterus will be found to be in good position and there will be no need for any abdominal operation to correct retroflexion. The final point to be decided in the treatment of patients near the menopause, whose prolapse is associated with bleeding, is whether vaginal hysterectomy should be performed before the plastic operation for prolapse or whether the uterus should be left and radium or X rays should be used to produce the menopause. The author prefers the latter course, because when hysterectomy has been performed the vagina must be so narrowed that coitus is impossible or difficult. In such instances domestic complications may ensue.

The Fruits of Conservatism.

VICTOR BONNEY (*The Journal of Obstetrics and Gynæcology of the British Empire*, March, 1937) states that he is impatient for a change of attitude of surgical procedure on the female organs, and he adds that, although many do lip service to conservatism, it is not practised by gynæcological surgeons in England or in other countries to the requisite extent. Amongst surgeons who occasionally operate on the female organs it is practised still less. An analysis of 632 myomectomies revealed a death rate of 1·1%; of 379 patients whom the author was able to follow up, new fibroids occurred in 2·37%, and menorrhagia either persisted after the operation or appeared subsequently in 3·1%. Amongst 379 patients who had myomectomy performed, 137 were within the child-bearing age; of these, 52, or 38%, conceived, there was one miscarriage, and Cæsarean section was performed 17 times. Several of the women had multiple pregnancies. As far as the author is aware, malignant diseases did not subsequently develop in the conserved uterus, except once, when a sarcomatous fibroid was removed without realization of the metamorphosis that had taken place. The author believes that, except in the case of malignant disease of the ovary, it is the duty of the surgeon to preserve the female sex organs intact or at least to leave the full amount of undiseased ovarian tissue that the patient possesses. He is of the opinion that there is no reason to remove the ovaries at the time of hysterectomy, and he states that the number of times that he has had to operate on ovaries conserved at hysterectomy can be counted on the fingers of one hand. To resort to radiological treatment of the ovaries for menorrhagia in women still in the thirties is both senseless and cruel. In respect to conservative treatment, there are many types of ovarian cysts which can be enucleated from the ovary; in this category are included dermoid cysts, follicular cysts, chocolate cyst. Reparative operations on the Fallopian tubes has been carried out 70 times, salpingostomy 44 times, freeing tubal kinks 7 times, tubal exsection and anastomosis twice, reimplantation into the uterus nine times, and once an ostium was made into the uterine corium after both tubes had been removed. Among 37 patients who replied to a *questionnaire* regarding pregnancy, seven, or 18%, had conceived, two after salpingostomy, two after reimplantation, one after reimplantation on one side and salpingostomy on the other side, one after double reimplantation with double salpingostomy, one after tubal exsection and anastomosis.

Cæsarean Hysterectomy.

G. VON BUD (*Monatsschrift für Geburtshülfe und Gynäkologie*, August, 1936) discusses the indications for hysterectomy during pregnancy, and describes his technique for the operation. The usual indications are accidental hæmorrhage, *placenta prævia*, atonic *post partum* hæmorrhage and the fear of infection. To these he adds two other indications: carcinoma of the cervix and the need for sterilization after repeated section for contracted pelvis. The pregnant uterus is brought outside the abdominal cavity, the round and broad ligaments are rapidly clamped and cut, the vesical fold of peritoneum is reflected and a subtotal hysterectomy is rapidly performed. The pregnant uterus is handed over to an assistant, who opens it and extracts the fœtus.

The Treatment of Uterine Hæmorrhage in Non-Malignant Diseases.

L. MARTINDALE (*The Journal of Obstetrics and Gynæcology of the British Empire*, March, 1937) compares the advantages of deep X ray therapy, radium and surgical procedures in the treatment of uterine hæmorrhage in non-malignant conditions. The author points out that before a decision is made regarding the best method of treatment, it is of primary importance to diagnose the cause of hæmorrhage and of the physical signs present, to know the age and civil state of the patient and the degree of which further child-bearing is important, and to have a full report of her general condition. The author concludes that deep X ray therapy is the method of choice in all uncomplicated cases of climacteric hæmorrhage due to *metropathia hæmorrhagica*, *fibrosis uteri* and small interstitial fibroids in which menorrhagia is the main symptom and the degree of anæmia is not so severe that the patient's life would be threatened if she were to have another severe menstrual period. Radium therapy should be chosen in cases of climacteric hæmorrhage due to the same causes, if the anæmia is severe enough to necessitate blood transfusion. Surgical treatment is indicated in all cases of doubtful diagnosis, and if the fibroid tumour

is larger than a four months' pregnancy; in all cases of subperitoneal and submucous fibroids and in cases complicated by neoplasms of ovaries or Fallopian tubes, or coexisting pregnancy. Abdominal myomectomy should be chosen for all young patients when possible, in order not to interfere with the possibility of a future pregnancy.

OBSTETRICS.

The Prevention of Puerperal Sepsis.

L. COLEBROOK (*The Journal of Obstetrics and Gynæcology of the British Empire*, August, 1936) gives his views as to the prevention of puerperal sepsis. As the result of ten years' experience, he considers that puerperal infection, both from the clinical and laboratory standpoint, may be divided into two broad categories: infection associated with injury to maternal tissue, and infection in which childbirth has been accompanied by little or no trauma. The ensuing puerperal fever in this latter type is attributable to the arrival from outside of the hæmolytic streptococcus. The author has no doubt that the organism is extragenital and that its advent could be prevented if prevention were set about in the right way. It is admitted that hæmolytic streptococci have been recovered from the genital tract of women before labour, but these are not generally of the pathogenic type; and as the result of the examination of swabs taken from eight hundred and fifty-five women, it may be stated that pathogenic hæmolytic streptococci are hardly ever present in the genital tract at the onset of labour. Nor does the author believe that the streptococci may be conveyed by way of the blood stream from such a focus as the tonsils to the genital tract. Pathogenic streptococci come most commonly from the throat, especially in tonsillitis, scarlet fever, *otitis media* and mastoid disease, erysipelas, nasal sinus infection, wound infection of all kinds, burns, whitlow, trivial finger infections and impetigo; lastly, symptoms resembling those of common cold or influenza may be associated with streptococcal infections. The author believes that about 7% of the population are carriers without having any apparent signs of definite infection. Another potential source which must be taken into account is the air. As regards prevention, an attempt should be made to detect hæmolytic streptococci among the whole of the midwifery personnel before they cause puerperal infection or disseminate streptococci, special attention being paid to those who have some obvious symptom. The next step is the immediate recognition of puerperal infection by the hæmolytic streptococci in order that further infection may be prevented. It is suggested that as soon as a rise of temperature to 101° F. occurs, a swab should be taken from the upper part of the vagina or cervix. Thirdly, every infective patient should be promptly removed to an isolation department.

Essential Hypertension and Eclampsia.

G. GAEHTGENS (*Monatsschrift für Geburtshülfe und Gynäkologie*, November, 1936) discusses the various theories regarding the effect of the toxin of eclampsia on renal function, especially in cases in which increased blood pressure is the single symptom. According to Seitz, about 3% of cases show this hypertension, which often is the only symptom manifested by women between twenty and thirty years of age. Increased tension is the cause and not the result of renal changes. The cause of the raised blood pressure is to be sought in the increased production of posterior pituitary hormone and its effect on the vegetative nervous system. Whenever the pregnancy is terminated the blood pressure rapidly falls to normal. If this does not occur, there is generally evidence of permanent renal damage. Therefore, cases should not be carried on too long when a raised blood pressure is a prominent symptom.

Rupture of the Symphysis During Labour.

H. HIRSCH (*Monatsschrift für Geburtshülfe und Gynäkologie*, November, 1936) discusses the ætiology of rupture of the *symphysis pubis*. The majority of reported cases followed some trauma during labour, and in only 15% to 20% did the condition occur spontaneously. The details of such a case are given. There had been an operation for sarcoma of the femur, but bony metatases were not found. The author proceeds to discuss the possible effect of hormonal influences in such cases. The work of Hisaw in describing a hormone "relaxin", contained in the *corpus luteum*, which produces separation of the symphysis in animals, is discussed; but there is no evidence that the same action occurs in the human being.

The Significance of Fœtal Heart Tones in Ablatio Placentæ.

G. C. RICHARDSON (*American Journal of Obstetrics and Gynecology*, September, 1936) makes a plea for the earlier diagnosis and prevention of fœtal mortality by careful observation of the fœtal heart tones in *ablatio placentæ*. Stormy as it may be, there is probably no condition in obstetrics which is so ceremonious in its course as *ablatio placentæ*. The events occur in an identical sequence without regard to the time factor involved, whether it be five minutes or five hours. The sequence is as follows: hæmorrhage, placental separation with increasing hæmorrhage, decreased oxygenation and increased fœtal carbon dioxide retention, fœtal embarrassment with compensatory acceleration of fœtal heart rate, fœtal heart compensation to maximum tolerance, asphyxia with decreasing fœtal heart rate, placental separation complete either before or after fœtal death, quiet or violent. The author suggests that action be governed by the fœtal condition. An early diagnosis is imperative and is made by directing attention to the fœtal conditions; unless delivery is imminent, immediate action should be taken. If action is taken upon the condition of the fœtal heart tones and not after they are gone, and if action is not deferred until the uterus is ligneous and if efforts are directed at saving the baby, the efforts on behalf of the mother will be more effectual.

Ectopic Pregnancy.

T. PÜTZ (*Monatsschrift für Geburtshülfe und Gynäkologie*, December, 1936) has analysed 300 cases of extrauterine pregnancy. The majority occurred in the tube and were equally divided between tubal abortion and tubal rupture. The average age of the patients was thirty-one years, and more than half had only one child or were sterile. Approximately seven years had elapsed between the previous pregnancy and the ectopic pregnancy. Evidence of previous inflammatory reactions in the appendages was obtained in many instances. The difficulty of diagnosing the condition before rupture is emphasized. The author considers that diagnosis is helped by estimation of the sedimentation rate, examination under "Evipan" anæsthesia, puncture of the posterior fornix, the Aschheim-Zondek test and exploratory laparotomy. The sedimentation rate is greatly increased, especially in cases of early rupture. Similarly, aspiration of the pouch of Douglas will settle the diagnosis in many doubtful cases. The author's mortality rate was 2% for the whole series.

Ætiology of Premature Separation of the Placenta.

K. HEIM (*Monatsschrift für Geburtshülfe und Gynäkologie*, December, 1936) has summarized modern research into the ætiology of accidental hæmorrhage and the toxæmias of pregnancy. He shows that in *ante partum* hæmorrhage, pre-eclampsia and eclampsia there is an increase in the amount of prolan secreted in the blood and urine. This fact is well known in the case of vesicular mole, but its importance as possibly the main ætiological factor in accidental hæmorrhage has not been previously noted. A similar rise in the prolan secretion was also observed in cases of hyperemesis and other toxic manifestations of early pregnancy. It would appear that the separation of the placenta is Nature's way of combating the excess prolan secretion, and indicates a gross lack of correlation between the hormones of the placenta and the *corpus luteum*.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held at the Renwick Hospital for Infants, Sydney, on November 19, 1936. The meeting took the form of a series of clinical demonstrations by the members of the honorary medical staff.

Erythrœdema.

DR. INGLIS ROBERTSON showed several patients who were suffering from erythrœdema or pink disease.

A baby girl, five months old, had been referred to the medical department on November 11, 1936, on account of unexplained photophobia of two weeks' duration. The child was irritable and refused food, and was constipated for the same period; its weight was stationary. On examination pronounced photophobia was found to be present, with moderately severe hypotonia. The hands and feet were pink, cold and clammy. There was no rash. The child had a clear watery nasal discharge. There was no cough, but the child was very irritable. The pulse rate was 180 in the minute (the record was taken when the child was struggling and crying). No teeth had erupted, but the child was worried with sore gums. The child was an only one. It was breast-fed; it had received orange juice. The child was suffering from the condition in an early stage.

A second child, aged nine and a half months, had a history of photophobia and irritability of six weeks' duration. The child had a brother who had also suffered from erythrœdema when twelve months old. The child's general condition was fairly good.

A boy, aged fifteen months, belonged to a family in which there were five other children, all of whom were normal. The child had been miserable and irritable for four months. It would not take its food and was losing weight. It would not sit up, but buried its head in the pillows; its hands and feet were cold and pink.

A child, aged ten months, had a history of being ill for one month. The mother first noticed a rash extending over the whole trunk; the child was also extremely irritable and restless. He had steadily lost weight and his muscles had become very soft. The mother said that the rash had become much worse and that the mouth had become severely ulcerated. On examination the child was found to be very ill. The lips and the mucous membranes of the cheeks were severely ulcerated. Hypotonia was present, with the puffy hands and peeling feet typical of the condition. The child sweated excessively. A severe rash involved the trunk and limbs. The pulse rate, when the child was at rest, was 166 in the minute. The child was unable to sleep either at night or during the day; it was eating well, and had but little photophobia.

Dr. Inglis Robertson explained that pink disease, when present in so severe a form as in the last patient, had not a good prognosis. The extreme restlessness, associated with a persistently rapid pulse rate and ulceration of the mouth or skin sepsis were a bad combination of symptoms. Happily this child had shown signs of improvement in the few days before the meeting—the pulse rate was becoming slower, the ulceration was ceasing to extend, the child was beginning to be able to sleep and was still taking its food well. Nausea or refusal of food was often a pronounced symptom, but this child had always taken its food well.

Retropharyngeal Abscess.

DR. MILTON COUTTS showed a baby, aged ten months, that was suffering from a retropharyngeal abscess. The baby was one of twins and had been admitted to hospital on November 1, 1936; its weight at birth had been six and a half pounds. The history of the present illness was that the child had suffered from anorexia for two weeks and a swelling under the chin for three days. The child had tended to choke at night, it could not swallow, and at times could not breathe properly. The respiration was "rattly". During a fit of coughing six days before the child's admission to hospital pieces of grass had been extracted from its nose. Examination revealed tenderness of the glands on the right side of the neck; the tonsils were normal, the pharynx was reddened, but no swelling was present. The erythrocytes numbered 3,620,000 per cubic millimetre, the hæmoglobin value was 70%, and the leucocytes numbered 10,000 per cubic millimetre. X ray examination revealed widening of the retropharyngeal space, and a diagnosis of retropharyngeal abscess was made. No foreign body was seen on X ray examination of the lungs.

On November 3, 1936, the child was drowsy and was not taking its food. A fluctuating swelling was present behind the right tonsil. A retropharyngeal abscess was opened with a knife and sinus forceps; gas anæsthesia and a sucker were used. The wound was probed every day for a week. The child gradually recovered.

Dr. Coutts explained that examination of a throat swabbing revealed no Klebs-Löffler bacilli; the pus contained *Staphylococcus aureus*. Dr. Coutts also pointed out that this acute primary type was met with between the third month and third year of life, 75% occurring in the first year of life.

DR. EDGAR STEPHEN asked where the abscess was opened and was it always a fatal disease if left unopened.

Dr. Coutts, in reply, said that it was the custom at the Renwick Hospital to administer a general anæsthetic (ether), then, with the patient's head bent back over the pillow, the tongue was depressed and an incision was made in the long axis of the body, in the most prominent part of the abscess, which was always on one side or the other (never in the mid-line); the most prominent part was at the level of the tonsil and usually presented in the oro-pharynx beside the tonsil. The incision was deliberately made small so that a sucker could readily remove all pus as it escaped; as the tension in the abscess decreased, the wound was enlarged with sinus forceps and the sucker was introduced into the cavity.

In reply to the second question, Dr. Coutts said that one baby had been admitted to hospital with a large undoubted retropharyngeal abscess and a history extending back two weeks. The parents had refused permission for operation, as in their opinion the child was getting better. When the child was seen three weeks later in the out-patient department all signs of the abscess had disappeared.

Laryngeal Stridor.

Dr. Coutts also showed a baby, aged two months, that was suffering from laryngeal stridor. The child had been admitted on October 2, 1936, when ten days old; at this time it weighed six pounds twelve ounces. The child was breast-fed and stridor had been present since birth.

On examination the child's general condition was good. Inspiratory stridor was present while the child was at rest, and the stridor increased in intensity on exertion or crying. The cry was normal. When the child was resting slight retraction of the soft tissues of the chest was noted, but when the child exerted itself the retraction became very pronounced. Examination of the chest revealed a resonant percussion note and vesicular breath sounds; there were no accompaniments. The throat was clear. Other systems were normal. There was no evidence of rickets. The lungs were poorly aerated. X ray examination revealed some atelectasis of the right lung. Direct laryngoscopy with Jackson's speculum and without an anæsthetic showed the epiglottis to be sharply folded so that the two sides were almost touching. The arytenoid cartilages were prolonged upwards and deviated medially and anteriorly, thereby approximating the arytenoepiglottic folds so that the upper aperture of the larynx was reduced to a long thin slit. The noise was made by the vibration of the soft folds during inspiration.

DR. COLIN VICKERY asked what was the prognosis.

Dr. Coutts, in reply, said that when death by suffocation did not occur in the early days of life, the condition

usually righted itself before the end of the second year. The greatest risk to life was an acute respiratory infection.

DR. GORDON LOWE asked the points of diagnosis from *laryngismus stridulus*.

Dr. Coutts, in reply, said that whereas congenital laryngeal stridor was a rare condition, *laryngismus stridulus* was comparatively common. This condition was usually limited to the first few weeks or months of life. The babies were usually of poor physique and many suffered from rickets. When the larynx was examined the epiglottis appeared to be quite devoid of cartilages and to be so soft that it was readily inhaled into the laryngeal aperture. This explained the peculiar sudden onset at the moment of walking or on commencing to cry. The child appeared to hold the breath and to remain in a state of suspended respiration until deeply cyanosed and unconscious. The first inspiration was usually accompanied by a loud stridor, but this did not persist.

Acute Mastoiditis.

Dr. Coutts's third patient was a child, aged eighteen months, who was shown in order to illustrate the routine treatment adopted at the hospital for acute mastoiditis. At the time of the child's admission to hospital the left ear had been discharging for six weeks, though very little pus had come away for two days; swelling had been present behind the ear for twenty-four hours. Examination revealed a thin purulent discharge coming from the left external auditory meatus, and œdema of the scalp over the mastoid, with pitting on pressure.

Operation was performed on August 4, 1936. An elliptical incision was made behind the ear; the periosteum was raised and the cribriform plate exposed. The mastoid was opened with gouge and mallet. The cells were found filled with pus and œdematous tissue. All the cells were removed with gouge and curette, and the bony posterior wall of the external canal was reduced with rongeur and chisel. The cavity was packed with inch ribbon gauze saturated with bismuth, iodoform and paraffin paste ("B.I.P.P."). The incision was closed with four interrupted silkworm gut sutures, the "B.I.P.P." gauze being left protruding from the lower angle of the wound. The external canal was packed with ribbon gauze.

On the day after operation the gauze was removed from the external canal and the wound was dressed every four hours with spirit and a dry dressing. On the fourth day after operation two inches of the gauze were removed from the wound; more gauze was removed every day. On the tenth day after operation the wound was healed, except at the lower end, and the sutures were removed. A small perforation was present in the inferior quadrant of the tympanic membrane; there was no discharge. Seven days later the perforation in the tympanic membrane was almost healed, the wound was healed, and the patient was discharged.

Dr. Coutts, in reply to Dr. Brodie Grant, said that it was customary to probe the lower angle of the wound daily after removal of the "B.I.P.P." gauze in order to drain away the serum, which, if allowed to accumulate, would force its way to the middle ear and retard the healing of the perforation in the tympanic membrane. The average time in hospital was less than three weeks. He had seen no recurrences.

Congenital Pyloric Stenosis.

DR. GORDON LOWE showed a patient, aged two months, who was suffering from congenital hypertrophic pyloric stenosis. The child had been admitted to hospital on October 13, 1936. It had been a healthy breast-fed baby until one month previously, when vomiting commenced. This gradually increased in severity, and at the time of the child's admission to hospital was projectile in type. The child was said to have lost five ounces in weight in the forty-eight hours prior to admission. It was constipated and had had no motion in the previous twenty-four hours. There was nothing of interest in the child's previous history nor in the family history.

Examination revealed a baby in fair general condition. The abdominal wall was very thin, no abdominal tumour was palpable, and "visible peristalsis" was present. The fontanelle was slightly depressed. The skin was lax and elastic, but somewhat dehydrated.

Operation was performed on October 13, 1936, and it was found that the pylorus was enlarged and irregular, very hard, and almost cartilaginous. Ramstedt's operation was performed.

After the operation brandy was given in ten-minim doses in water every two hours. Glucose was given in a 5% saline solution *per rectum* every four hours. Half-normal saline was given in sips between glucose feeds, up to as much as two teaspoonfuls. Breast feeding was recommenced on October 15 and was supplemented with whey. On October 17 pallor had developed. When a blood count was taken, it was found that the red cells numbered 3,500,000 per cubic millimetre, the hæmoglobin value was 65%, and the colour index 0·9. The leucocytes numbered 7,000 per cubic millimetre.

On October 19 the sutures were removed. In the first two weeks there was a loss of weight of two pounds; in the third week there was a gain of ten ounces. There was occasional vomiting for the first nine days after the operation, and mucus was found in the stools for four days. The child was discharged from hospital, cured, on November 5. There was no vomiting, the stools were normal, and weight was increasing.

Dr. Lowe also showed a patient, aged seven weeks, who was suffering from congenital pyloric stenosis. The child was admitted to hospital on September 6 with a previous history of rectal hæmorrhage and hæmatemesis twenty-four hours after birth. Three weeks prior to admission it began to vomit once a day after food. This continued until four days before the child's admission to hospital, when it was taken off the breast and was given "Vi-lactogen". Vomiting then occurred one hour after each meal. Vomiting was projectile in type from the beginning.

Physical examination revealed a child in fair general condition. The abdomen was slightly distended, and a small mass was palpated deeply in the pyloric region. The tissues were lax and dehydrated. A test feed of four ounces of a 10% glucose solution was given, and definite peristaltic waves were seen. Projectile vomiting soon followed.

Ramstedt's operation was performed on September 6 and a hard tumour of the pylorus was discovered. After the operation one-ounce feeds of 5% glucose in saline solution were given *per rectum* every four hours. Half-normal saline solution was given between glucose feeds, up to one ounce. On September 8 expressed mother's milk feeding was reintroduced. On September 14 a small stitch abscess had developed. On September 15 all sutures were removed. There was occasional vomiting for the first four days after the operation. There was practically no variation in weight while the child was in hospital, and it was discharged, cured, on September 24, weighing seven pounds six ounces.

Dr. Lowe pointed out that rectal hæmorrhage and hæmatemesis when the child was twenty-four hours old, with pyloric stenosis later, suggested the possibility of a congenital pyloric ulcer. The patient was followed up. On October 1 the child's weight was seven pounds fifteen ounces; on October 8, eight pounds nine ounces; on October 26, nine pounds fifteen ounces; on November 5, ten pounds eight ounces. After that the child was considered to be doing well.

Osteomyelitis of the Jaw.

Dr. Lowe's third patient was a girl, aged one month, who was suffering from osteomyelitis of the jaw. The child was admitted to hospital on December 12, 1935, with a history of a lump in the left side of the face from the age of eight days. This lump was increasing in size. Physical examination revealed a fluctuant mass in the left parotid region with an abscess point in the mouth. This point was opened and the opening was enlarged with sinus forceps and a small quantity of pus escaped. On December 3, twenty cubic centimetres of thick gelatinous pus were

aspirated. On December 6 an incision was made over the pointing area on the chin. On December 22 an X ray examination was made. The radiologist reported evidence of osteomyelitis of the left horizontal ramus and of the angle of the jaw. Staphylococci and pneumococci were found in the pus. On January 11, 1936, the child was discharged from hospital relieved.

The patient was followed up, and X ray photographs were taken on five subsequent dates. On February 19, 1936, the photograph showed a typical osteomyelitis with no free sequestrum; on May 25 the general appearance was one of resolution. On September 3 X ray examination showed that irregularity and erosion were still present on the left side of the mandible; there was no gross sequestrum. On October 20 the sinus was curetted and the sequestrum was removed.

(To be continued.)

MEDICO-POLITICAL.

A MEETING of the Victorian Branch of the British Medical Association was held at the Medical Society Hall, Albert Street, East Melbourne, on April 7, 1937, PROFESSOR R. MARSHALL ALLAN, the President, in the chair.

Workers' Compensation in Victoria.

DR. C. H. DICKSON, Medical Secretary of the Victorian Branch, opened a discussion on workers' compensation in Victoria. He said that the history of workers' compensation legislation in Victoria commenced in 1908. In that year, and annually until 1914, bills had been introduced in the State House of Parliament; but it was not until 1915 that the first act was proclaimed. Amending acts had come into force in 1922 and 1928, and in the latter year the *Workers' Compensation Acts* were consolidated in a measure which had remained unchanged until the passage of an amending act in 1935 (proclaimed in 1936) and a further amending act in 1936, which had been proclaimed on March 15, 1937.

With every reopening of the act, said Dr. Dickson, its provisions had been liberalized in various directions: the income limit of a "worker" had been raised; the period of disability before compensation could be claimed had been lessened; death benefits and weekly payments had been increased; the list of occupational diseases extended; and so on. But it was not until 1935 that any provision, with the exception of a minor one which would be mentioned later, for the payment of the ambulance, medical and hospital expenses of injured workers had been included.

Dr. Dickson pointed out that benefits under the acts at the present time extended to all employed persons except, in the first place, a person whose remuneration exceeded £400 a year; in the second place, a person whose employment was casual and who was employed otherwise than for the purposes of the employer's trade or business; in the third place, a member of the employer's family dwelling in his house.

It was obligatory for all employers to insure their employees, and those who failed to do so were liable to heavy penalties. In the past the penalties were light; consequently certain unscrupulous employers had found that it was cheaper to risk prosecution for failure to insure their employees rather than to pay the necessary premiums. The result had been, of course, that cases of hardship had occurred when the injured employee or his dependants sought to recover compensation and found the employer to be a "man of straw".

Speaking of the amounts of compensation payable at the present time, Dr. Dickson said that they were quite substantial. First, when dependants totally dependent on the worker's earnings were left, a sum was allowed equal to four years' earnings, the minimum being £400 and the maximum £750. Secondly, when dependants in part dependent on the worker's earnings were left, an amount agreed upon or determined by arbitration was allowed. Thirdly, when no dependants were left, the reasonable expenses of medical attendance, maintenance in hospital and burial, up to a maximum of £75, were allowed.

He pointed out that the last proviso was interesting. It had been in the act for many years, but did not appear to have been known to members of the medical profession. In most of the cases coming under the provision, the patients had, of course, been treated in public hospitals by honorary medical officers, with the result that the insurance companies had been relieved of any expense for medical attendance. To ensure the medical service's being paid for in such cases in the future honorary medical officers were being urged to persuade their hospital committees to seek authority from the Charities Board to declare any bed a "pay-bed". Provision for the granting of this authority existed in the regulations of the *Hospitals and Charities Act*. If this suggestion was adopted, medical fees for service to such cases could be claimed.

Dr. Dickson said that in the case of permanent injuries, such as the total loss of sight or of a limb or part thereof, as specified in the fourth schedule to the acts, lump sums up to £750 were payable, in addition to weekly compensation, during temporary total incapacity, to a maximum of £25. When temporary total incapacity for work followed an accident to a worker, he was entitled to weekly compensation ranging from £1 per week (for a worker under twenty-one years of age) to £3 7s. 6d. per week for a worker with four or more children under fourteen years of age dependent on him.

In computing the amount of compensation payable under the acts, Dr. Dickson pointed out that the employer was debarred from taking into consideration any sum due to the worker from another source, such as sick pay from a lodge or benefits under an assurance policy.

One curious anomaly had been perpetuated in the acts. It was not clear why the loss of the whole or portion of the right upper extremity should be regarded as more serious than a similar loss of the left upper extremity; but it was a fact that loss of the whole of the right arm was valued at £600, while loss of the left was valued at only £562 10s., with comparable differentiations for less severe injuries. To one who was left-handed it appeared to be an invidious distinction.

Dr. Dickson said that that concluded his summary of the worker's side of the acts. The next question was, what benefits did the doctor receive? The answer was that until recently he had received nothing, and Dr. Dickson supposed that it had been the experience of most of those present, who had attended such cases in the past, to be cheerfully told by the patient: "Of course, doctor, I'm insured", and for the patient to be quite oblivious of the fact that no medical benefit was provided; so they, as doctors, had added service in such cases to the long list of involuntary honorary appointments already held for service to sustenance workers, old-age pensioners *et cetera*. The principle of payment for medical services to workers coming under the provision of workers' compensation acts had, of course, been accepted in less backward countries for many years, and in New South Wales and Western Australia adequate sums to cover medical and hospital expenses had been provided.

The 1935 bill had no reference to medical expenses of injured workers; but at the last minute amendments in the House had included a section granting, in addition to other compensation payable, the sum of £5 to cover the ambulance, medical and hospital expenses of injured workers, in that order of priority of claim.

Dr. Dickson then quoted the following section relating to medical expenses:

> Any fees payable to any medical practitioner in respect of any medical or surgical treatment or examination arising immediately out of the accident.

He said that shortly after the passing of the act the State Insurance Commissioner had informed him that he had based his premium increase on an estimated average claim of £1 11s. 6d. for medical services in such cases. At the same time he had issued a circular setting out the benefits provided by the new act, which included the

statements: "Payment up to £5 of fees to medical practitioners in respect of medical or surgical treatment" and "medical benefits up to £5".

The act had come into force on March 2, 1936, and soon after that date the office was deluged with letters from members complaining that the insurers were refusing to pay more than 10s. 6d. for the treatment of any one patient. To any normal person the words "arising immediately out of the accident" meant "directly due to" the accident, but somebody in the insurance world had had a bright idea, and with the aid of legal opinion had decided that "immediately" meant "instantly", "at once", and on that basis had disclaimed liability for anything more than the first visit of the doctor. Some most amazing distortions of the wording of the act had been sent by insurance companies in letters to doctors and employers. Dr. Dickson quoted the following examples:

> The act provides only for fees being paid to medical practitioners for the initial medical or surgical treatment.
>
> This company insures Mr. ..... liability towards employees under the above acts, and 10s. 6d. is the limit of our client's liability for medical expenses.
>
> The employer is liable only for the first or emergency treatment.
>
> The recent revision of the act provided for payment for certain medical expenses where a doctor had to be called in immediately.
>
> The employer is liable to pay medical expenses incurred for urgent immediate medical treatment.
>
> The act does not make an employer liable to pay medical fees, except the initial charge if incurred immediately after an accident.

One company, which, Dr. Dickson thought, could be described as really "hard-boiled", had stated that: "No liability attaches for medical fees under the Victorian *Workers' Compensation Act*."

During all this time great stress had been laid on the opinion of an "eminent King's Counsel" as supporting the point of view of the insurance company. At the same time another equally eminent King's Counsel had used the following words to the insurance companies:

> I have reached the opinion that the word "immediately" has no reference to time, but is used as requiring a certain directness of causal connexion between the accident and the medical of surgical treatment or examination. It is intended, I think, to exclude treatment which is only remotely or indirectly occasioned by the accident.
>
> If the legislature had intended to refer only to first aid, or some original emergency treatment, I think it would have said so in plainer words.

In succeeding months as much publicity as possible had been sought by the Association, said Dr. Dickson. The interpretation of the insurers had been brought to the notice of the Premier at a deputation in June, and he had stated that it was not in accord with the intentions of Parliament. Questions had been asked in the House, members of the Branch had been circularized, and the insurance companies had been badgered by having their point of view challenged on every possible occasion.

The result was that when an amending bill was introduced in December, 1936, the word "immediately" had been deleted, the amount of compensation increased from £5 to £10, and the definition of a hospital extended to include not only an "institution" within the meaning of the *Hospitals and Charities Act* (in other words, a public hospital), but also "any hospital registered under the *Health Act* 1928", which meant the inclusion of all private, intermediate and bush nursing hospitals as hospitals in which a worker could be treated. It had been possible, said Dr. Dickson, to have the words "any hospital registered under the *Health Act*" inserted in place of a most extraordinary clause in the bill, which referred to hospitals within a certain radius of the worker's home or the scene of the accident, the acceptance of which would have led to endless confusion. Dr. Dickson then quoted the section as it read at the present time:

> The employer shall not be liable under this Act in respect of any injury which does not disable the worker for a period of at least three days from earning full wages at the work at which he was employed.
>
> Where an employer is liable to pay compensation in respect of any personal injury by accident to a worker arising out of and in the course of the employment of that worker, there shall in addition to any compensation otherwise payable under the *Workers' Compensation Acts* be paid by or on behalf of the employer in the following order of priority:
>
> (i) Any expenses for ambulance services necessarily arising out of the accident.
>
> (ii) Any fees payable to any medical practitioner in respect of any medical or surgical treatment or examination arising out of the accident; and
>
> (iii) Any expenses reasonably incurred by any hospital in affording treatment arising out of the accident to a worker;
>
> Provided that the amount required to be paid under this Sub-Section shall not exceed ten pounds.
>
> In this Section "Hospital" means "Institution" within the meaning of the *Hospitals and Charities Act* 1928 at which medical or surgical treatment for in-patients and out-patients is provided and any hospital registered under the *Health Act* 1928.

It was difficult to see any way in which the insurers' responsibilities in regard to medical expenses could be evaded; but there were one or two other points that might cause argument.

The words "any expenses reasonably incurred by any hospital" were applicable when the scope of the section was limited to a public hospital; but their meaning when applied to a private hospital was open to discussion. A consequential amendment should have been made when the subclause was altered to include private hospitals; but, although a suitable draft amendment was submitted to members of Parliament, the matter had apparently been overlooked. Further, Dr. Dickson asked, what was an "ambulance"? In the Legislative Council on December 16, 1936, the Honourable M. Saltau had stated:

> A case was brought under my notice concerning a young man who met with a very serious accident through falling off a motor cycle in the Otway Forest. He was ordered to be taken to hospital as quickly as possible, but no ambulance was available, the nearest being at the Colac Hospital, to which he was eventually taken. A motor car brought from Beech Forest conveyed the young man to hospital in half the time it would have taken if the ambulance had been requisitioned, and at less than half the cost, but the State Accident Insurance Office would not recognize the expenditure because the injured man was not conveyed in an ordinary ambulance.

The word "ambulance" was not defined in the Act; but Dr. Dickson quoted the "Shorter Oxford English Dictionary's" definition:

> (1) A moving hospital, attending an army as it moves, so as to succour the wounded without delay;
>
> (2) An ambulance wagon or cart for conveying the wounded off the field, etc.

Refusal to pay in the case quoted seemed, therefore, quite indefensible.

What was meant by the provision for payment "in the following order of priority" of ambulance, medical and hospital expenses? Counsel's opinion could again be quoted as follows:

> A second question is also submitted to me:
>
> If a worker incurs emergency hospital expenses, after which he obtains the services of an ambulance and a medical practitioner, is he entitled to be paid for the latter services?
>
> The answer to this question must, in my opinion, be in the affirmative. The priority prescribed by Section 4 (1) has no reference, as I think, to the

order of time in which the different services are given to the worker. The priority prescribed is a priority of right to payment. Thus, if the cost of ambulance services were £2, of medical services £2, and of hospital services £2, the result would be that the ambulance services would be paid in full and medical fees would be paid in full, whereas the hospital would receive only £1, since the maximum required to be paid by the subsection is £5 in all. This would be so, in my opinion, whatever might be the order in which the worker had occasion to avail himself of the three kinds of services mentioned.

This opinion, Dr. Dickson said, had been given when a sum of £5 and not £10 was provided in the act. However, so far as he was aware, no dispute regarding rights to priority of payment had yet arisen.

Two other important problems remained to be settled: first, the position of lodge patients coming under the provisions of the act, and secondly, the question of the removal from the public hospitals of as many patients benefiting from the act as possible.

Speaking of the position in regard to lodge patients, Dr. Dickson said that he would quote the wording of the lodge agreement, which was:

> But if the member . . . recovers compensation only under the *Workers' Compensation Act*, 1915, the medical officer shall accept the rates of payment set out in Clause 8 hereof as a full discharge of the member's liability to him for such services.

They were advised legally, he said, that under the *Acts Interpretation Act* the phrase *"Workers' Compensation Act*, 1915" included subsequent acts. It would be remembered that the relevant clause in the act provided that the injured worker could claim from his employer reimbursement of "any fees payable to any medical practitioner in respect of any medical or surgical treatment or examination arising out of the accident". It would therefore be seen that for the capitation fee the lodge medical officer had agreed to treat the lodge member, and as the latter did not incur any liability for fees he therefore had no claim under the act; but if the lodge member incurred fees under the provisions of Clause 6 of the agreement, he did incur liability and could claim reimbursement from his employer. If, for example, a lodge patient sustained an injury not necessitating anæsthetic and operation, the medical officer had no claim for fees; but if an operation and anæsthetic followed the accident, the medical officer could claim fees, which in turn the member could claim from his employer. They had been aware of this aspect for some time, Dr. Dickson continued, and negotiations had been proceeding with the lodges for the past six months. Finality had practically been reached and the lodge agreement would be altered in terms somewhat similar to those he would now quote:

> Provided lastly that if a member sustains personal injury in respect of which he has a claim for compensation under any of the acts referred to in the last preceding proviso or any subsisting amendment thereof the medical officer shall be entitled to claim and receive from such member in addition to the fees chargeable by him under the provisions of Clause 6 hereof fair and reasonable remuneration for professional services rendered to such member in respect of such injury but such member shall not be required to pay to the medical officer for services other than those referred to in Clause 6 thereof any sum greater than the amount received by him for medical expenses under the provisions of any of the said acts or any subsisting amendment thereof.

A similar alteration had been made in the agreements in Western Australia and New South Wales when the *Compensation Acts* came into force in those States.

The insurers, he said, were alive to the fact that the lodge agreement debarred medical officers from claiming fees in certain cases and were now asking all claimants for compensation whether or not they were lodge members. It was superfluous to point out that the insurance companies had not computed the employer's premium on the basis that certain employees were entitled to lodge benefits; but for the present doctors could only accept the situation, and members of the Branch were assured that everything possible was being done to rectify the position. Actually lodge agreements were made between the individual medical officer and the trustees of each friendly society, and it would be necessary to have several thousand supplementary agreements printed and signed by the parties concerned as soon as negotiations with the lodges were completed.

The second important matter, said Dr. Dickson, was to ensure the removal of as many patients as possible from the public hospitals. He went on to quote an extract from the "British Hospitals Year Book 1936", which, he thought, applied to the existing position in Victoria:

> Too long it has been fashionable to make use of the existing voluntary hospitals as industrial and even governmental stand-bys. Voluntary effort is properly employed in looking after those who cannot pay for what they need and not in shouldering the responsibilities of those who can and who would have to do so were there no voluntary hospitals. To expect voluntary effort to undertake financial responsibilities for the treatment of what are mainly industrial burdens is to add insult to injury. The prompt treatment of the victim relieves the pockets of the employer, of the mutual insurers, of the friendly societies and of the rates. Why voluntary effort should be expected to pay one penny of the cost baffles imagination.

Records kept in 1935 showed that in a period of six weeks at the Royal Melbourne Hospital alone 611 separate victims of industrial accidents had received out-patient treatment and 18 in-patients had occupied 256 bed-days, at a cost to the hospital of £118 1s. 6d. It might have been expected that the hospitals would have welcomed a compensation act which enabled an injured worker to obtain private medical attention and thus relieve the hospital of some of its burden; but, instead of that, the hospitals looked on the act as a means of increasing their revenue. In some hospitals special clerks were employed to keep records of the cases, and there was considerable jubilation in one institution, which estimated that the act would add £1,500 a year to its income. Many hospitals went further and actually made agreements with insurance companies to treat patients coming under the provisions of the act for fixed sums: 3s. a day for out-patients and 9s. a day for in-patients. Counsel's opinion on the right of a public hospital committee to make such agreements had been obtained; but, as that opinion had not yet been considered by the Branch Council, Dr. Dickson was not at liberty to disclose it. He was glad to learn that one honorary surgeon had, within the last fortnight, taken the matter into his own hands and turned compensation patients away from his clinic. Obviously doctors could not refuse to treat all patients coming under the act at public hospitals, as severe injuries would not be covered by the £10 available. Only on the day of the meeting, however, had Dr. Dickson heard that one hospital, which was reluctant to part with any of its out-patients, was raising the question of who was to decide, when the injured worker first attended the hospital, whether he would be incapacitated for three days and so come under the provisions of the act. If this decision could not be made there was no justification for refusing treatment. The answer to such an argument was obvious. Practically all patients could be turned away, as only employed persons came within the scope of the act, and if the injury was not severe enough to warrant absence from work for three days, the liability for medical expenses would be small and should be capable of being met by an employed person; if the injury was sufficiently serious to entail absence from work for three days, the worker would come under the provisions of the act.

Study of the acts showed that Parliament had sought to protect the interests of the worker as much as possible and to safeguard his rights. For that reason choice of

doctor had not been restricted and the following points should be noted:

First, a "certifying medical practitioner" was a doctor appointed as such by the Governor-in-Council under the *Workers' Compensation Act* or the *Factories and Shops Act*. Certificates dealing with industrial diseases (poisoning by heavy metals, anthrax *et cetera*, as specified in the fifth schedule), or with aged and infirm workers, were valid only if signed by a certifying medical practitioner. The certificate of any legally qualified medical practitioner was valid in any case of injury sustained by a worker in the course of his employment.

Secondly, the act gave the employer the right to require the injured worker to submit himself for examination by a doctor provided by the employer. Certain minor restrictions of the employer's right concerning the frequency and time of attendance were imposed by the regulations, Part iii. There was no provision in the acts or regulations giving any greater weight or authority to the opinion of the practitioner representing the employer, or the insurance company, than to that of the practitioner representing the worker, and the common belief that the employer's doctor was a medical referee in these cases was quite unwarranted. Neither party to a claim under the act need accept the view of the medical adviser of the other, and, if agreement could not be arrived at, all questions as to liability to pay compensation and as to the amount or duration of the compensation must be settled by arbitration. The only provisions of the acts giving any authority to the decision of any person other than an arbitrator were contained in those sections, which Dr. Dickson would quote, dealing with:

> (*a*) Cases where a claimant is aggrieved by the refusal of a certifying medical practitioner to give a certificate of disablement from industrial disease, or the employer is aggrieved by the giving of such certificate. In such cases the question of the claimant's disablement may be referred to a medical referee, whose decision is final.
>
> (*b*) Cases where the question of the extent of disablement, either from accident or industrial disease, is in dispute may be referred to a medical referee for final decision, provided both employer and employee agree to such method of settling the dispute.

Dr. Dickson said that the medical referee in cases under (*a*) or (*b*) was a doctor appointed as such by the Governor-in-Council, and was distinct from the doctor employed by either party to a dispute.

What, it would be asked, were the prospects of the introduction of a more adequate act in Victoria? Probably they were good. Labour was determined to have a board, established on the lines of the Workers' Compensation Commission of New South Wales, to which all claims would be referred. It was alleged that injured employees were at times defrauded by unscrupulous insurance companies and bluffed into accepting less compensation than that to which they were entitled. The fact that all settlements were subject to the approval of such a board would prevent sharp practice.

If increased medical benefits were to be obtained, and they were necessary, the profession must seek them with clean hands, and that was a matter to which Dr. Norris would refer; but, apart from the adoption of a high standard of professional conduct, every member of the Branch could personally assist in seeking increased benefits. Dr. Dickson closed with the reminder that individual propaganda would be of the utmost value.

DR. F. KINGSLEY NORRIS said that the Medical Secretary had told those present something of the improvements that had been brought about by alterations in recent *Workers' Compensation Acts*. That they as a profession had benefited by these recent acts was due almost entirely to the industry of one man—their Medical Secretary, Dr. Dickson—and was another example of the fact that the welfare of the general practitioner was not solely in the hands of men in general practice, as was so often suggested in that room.

They had heard of some of the Association's difficulties with the insurance companies. The next question was, what were the insurance companies' difficulties with the Association? Of what did they complain? The answer was, mainly of three things: first, careless and false certification; second, the charging of fees out of proportion to the services rendered; third, the failure of doctors to recognize their limitations, and by their neglect to seek or advise specialist assistance, their delaying of the recovery of injured workers and mulcting the insurance companies of excessive amounts of compensation by prolonging the patient's disability.

With regard to careless and false certification, Dr. Norris said that it would be remembered that in 1936 a circular dealing with medical certificates was sent to all members of the Branch, and he did not wish to enlarge on the subject at the moment; but it was distressing to be told by one of their members, who was associated with insurance companies, that whereas ten years ago, when he visited an insurance office to advise on claims, the manager used to say: "We need not worry about this case, there's a medical certificate with it"; the manager nowadays would say: "Is the medical certificate with this case of any value?"

They might have read in THE MEDICAL JOURNAL OF AUSTRALIA of September 12, 1936, an address by the retiring president of the Western Australian Branch, Dr. F. L. Gill. If they had not read it, Dr. Norris suggested that they do so. Dr. Gill, in his address, had discussed some of the difficulties in workers' compensation matters in that State, and had referred particularly to the doctors who rendered excessive accounts. In Western Australia and New South Wales a committee of representatives of the British Medical Association and the underwriters had been established to deal with disputed accounts, and when, during the debate on the 1936 bill, the profession in Victoria was accused in their own House of being likely to charge unreasonable fees if the amount available was increased to more than £5, several members of Parliament were informed that if an adequate amount for medical service was provided this Branch would be willing to appoint a similar committee.

Dr. Norris said that some unworthy cases had occurred in Western Australia. Dr. Gill had quoted one of them:

> The worker was employed on road construction and received a wound on the leg. He was in hospital for seventy-two days, was certified fit for work on the seventy-third day, and the doctor charged for seventy-three visits, including one per day up to the last day.

How could such conduct be justified, Dr. Norris asked. False certification was included in the definition of infamous conduct issued by the Medical Board of Victoria, and as such rendered a medical man liable to deregistration.

Coming to the question of excessive fees, Dr. Norris said that the complaint of the insurers in other States, that doctors charged fees in compensation cases which would be above the ability of the average wage-earner to pay if he were not insured, was, of course, untenable. Insurance was provided so that adequate fees could be paid; but there was no excuse for any doctor claiming £10 for a service which he well know was worth only £5.

Incidentally, whenever the question of increased medical benefits was raised in Parliament, he said, the old cry of the burden on industry was heard. Examination of the position in New South Wales, where a sum of £25 was provided for medical expenses, showed that this was a fallacy. Dr. Norris quoted the statistics of the Workers' Compensation Commission for the year 1934-1935:

Wages paid to insured workers	£107,563,366
Premium income	£1,168,498
Percentage of premium to wages	1·09%

A sum of £93,762 had been paid for medical treatment, representing 14% of the total paid in compensation, under 8% of the total premiums paid, and a negligible percentage of the total wages of the insured workers.

The remedy for the last complaint of the insurers, namely, failure to recognize limitations, was obvious. No doctor need be ashamed to seek the help of his colleagues in difficult cases, and he should not hesitate to do so.

Finally, therefore, Dr. Norris appealed to all members of the Branch to conduct their practices in such a way that no stones could be thrown when improvements in the *Workers' Compensation Act* were sought.

DR. W. H. FITCHETT asked what provision there was in the act for an allocation of the medical fee paid for services to an injured worker when the services involved those of a medical practitioner and a radiologist or other specialist.

Dr. Dickson, in reply, said that the provision in the act read: "any fees payable to any practitioner in respect of any medical or surgical treatment or examination arising out of the accident". The words "or examination" would include X ray examination.

Dr. Fitchett suggested a hypothetical case, in which £8 were left of the £10, the ambulance expenses having been £2. If the cost of the medical attention was £20, including an amount of £4 due to the radiologist, he asked how much of the £8 would be paid to the radiologist.

Dr. Dickson replied that the act contained no provision for specific payments for the different services required in the medical treatment. In the case stated £8 would be provided under the act towards the cost of the treatment. For the balance (£12) the injured worker would be liable personally. Under the New South Wales act the position was somewhat different. A sum of £25 was provided for medical fees and £25 for hospital fees. Medical fees for various treatments were provided under a schedule, and although that schedule was not binding on the doctor who thought himself entitled to higher fees, the New South Wales Branch discouraged the practice of claiming fees higher than those set out in the schedule. There was this point, too, to remember: if the fees to be charged were higher than the worker could afford, he could claim the right of treatment in a public hospital.

DR. DAVID ROBERT said that one of the possibilities mentioned by Dr. Norris, if the profession did not give faithful service, was that the insurance companies would pick out certain doctors who would undertake all the necessary treatment for injured workers. The point he wished to make was this: even if the profession did its share, what was to prevent the insurance companies from appointing certain doctors to do the whole of the work, no matter how low the profession's charges were? No matter what fees were charged, the profession was bound to be faced with the position of the appointment by the insurance companies of certain doctors to effect throughout the State the treatment required under the provisions of the *Workers' Compensation Act*.

DR. JOHN KENNEDY said that the late Sir William McPherson, when he was Premier, realized that industry was not carrying its weight in the matter of the injured worker. He took up the challenge, but unfortunately was relieved of office before he had achieved anything. Victoria was now realizing its responsibility, and the time was not far distant when the conditions existing in New South Wales would also prevail in Victoria. With regard to the reference to fraudulent settlements made by insurance companies, perhaps it was not generally known that any settlement made between an insurance company and an injured worker must be submitted to the Registrar before being completed. The Registrar would refuse to recognize any agreement reached fraudulently, and therefore it would have no legal effect. Insurance companies, since the amendment of the act, were giving consideration to the treatment of injured workers. The company with which he (Dr. Kennedy) was connected, covered about a third of the accident insurance business of the State. The accidents in which it was interested numbered about 15,000 a year. A little while ago that company had thought of establishing a special centre for treatment of the injured workers insured by it in the metropolitan area. If that project was gone on with, the profession would be faced with a problem. Dr. Kennedy wondered what the reaction of the medical profession would be to such a centre. There was a section of the profession that had adopted the practice of sending every worker, whose injury required treatment calling for just ordinary skill, to the hospitals. Another company, which was dissatisfied with the treatment given by certain practitioners, had selected men who had done good work, and indicated to the employers that those were the doctors to whom injured workers should be taken in case of accident or injury. The whole matter was in the hands of the general practitioner. If he did his work well and his charges were reasonable, there would be no special centre; but if his work was not well done or his charges were excessive, there would be. The Council and members of the Branch were not aware of the extent to which bad certification was going on. The certification was not so much false as really foolish—certification that could not be based on either anatomical or pathological grounds. As a matter of fact, the certification was so extraordinary that the insurance companies took no notice whatever of the certificates provided by the great mass of the men in medical practice.

DR. SYDNEY CRAWCOUR said he wondered whether it was within the powers of the insurance companies to establish such centres as suggested by Dr. Kennedy. The act allowed the employee a certain sum of money to defray his medical expenses, and it could be assumed that the employee had the right to seek the medical advice he needed, wherever he chose to secure it. By what authority could the employer or the insurance company insist upon an injured worker being treated at a particular centre? With regard to the accusations against insurance companies, he had to admit that since the act had been operating any claim he had presented had been paid in full. Apropos of insurance claims, there was a doubt as to whether insurers were liable for certain accidents and injuries. There was hernia, for instance. The types of accidents and injuries for which insurers were liable was a matter of great interest, and perhaps it could be defined at a later date. Then there were the questions of responsibility for the fee, and of the person to whom the account was to be rendered. It was hard to see how the lodge agreement had any bearing on payment if under the act the insurance company was directly responsible for the payment. Finally, there was the question of the payment for certificates. If a patient came along for treatment and during that treatment required a certain certificate, what charge had to be made for that certificate, and to whom must the account be rendered?

Dr. Dickson, in reply to Dr. Crawcour, said that under the act as it stood at present the insurer or the employer had no power to compel an injured worker to receive treatment at any particular place. The patient had entirely free choice as to where or by whom he would be treated. On the question of liability in respect of treatment for hernia, the position was now fairly well defined. In the last report of the New South Wales Insurance Commissioners there was a lengthy article by Dr. Mackay. With regard to the responsibility for payments, the act stated: "That where an employer is liable to pay compensation . . . there shall be in addition to any compensation otherwise payable . . . any fees payable to any medical practitioner." Therefore to comply strictly with the provisions of the act the account for medical services should be rendered to the patient. The fees then became payable to the medical practitioner, the patient including the medical fees in his claim for compensation. In practice, however, the insurance companies had paid the accounts directly to the doctor. Whether they would continue to do that remained to be seen. It was likely that the companies would prefer to deal directly with the doctor. Otherwise the patient might collect the money and not pay the doctor. As to charging for certificates under the existing acts, the insurance companies issued forms in which it was stated: "No fee is payable for this certificate by the company"; there was still no provision for payment for it, but as there was an amount of £10 available for medical attention, it did not really matter. The patient could either be made to pay for the certificate himself or the certificate could be thrown in with the treatment. The only people entitled to statutory fees for certificates were the medical referees, who were medical practitioners appointed to adjudicate in cases in dispute, and medical practitioners sitting in court as assessors, and "certifying medical practitioners".

Dr. Norris, in reply to Dr. Roseby, said that the reasons for the opening of treatment centres by insurance companies were those stated by Dr. Kennedy—unsuccessful treatment of injured employees and unreasonable charges for treatment by the profession. The companies had already flown one or two kites with a view to getting the work done cheaply. The profession, through the Association, could definitely control one situation should it arise, but the question of successful or unsuccessful treatment was one that rested with the profession itself. The general practitioner should be able to give treatment just as skilled as that which could be obtained at a central clinic. But if the skill required for successful treatment was beyond the practitioner, he should not hesitate to enlist it elsewhere.

Dr. Kennedy said that one insurance company in New South Wales already owned quite a large centre for dealing with injured workers, and in it some 200 patients were treated every day.

DR. KENNETH SMITH suggested that the lodge agreement be altered by the insertion of the word "disease" after "accident". Compensation should be paid for treatment of disease arising out of employment as well as injury arising from an accident during employment.

DR. S. CRAWCOUR asked whether it was necessary to have the patient's permission before sending a report to an insurance company.

Dr. Dickson replied that as payment depended upon a claim following a report, it could be taken that no objection would be raised by the patient as to the reporting of the injury on which he was claiming compensation.

DR. A. E. COATES, on behalf of the Science Subcommittee, thanked Dr. Dickson and Dr. Norris for the interesting addresses.

From the attendance and interest shown in the meeting it appeared that there was a demand for more addresses on similar matters. The Science Subcommittee would bear it in mind and from time to time ask other members to speak on other matters dealing with industrial medicine.

DR. J. A. CAHILL pointed out that through the action of the insurance companies the convalescence of many patients was delayed. Some companies insisted on the patients going into the city for examination when they should be resting at home in bed.

PROFESSOR R. MARSHALL ALLAN referred to the tremendous amount of work done by Dr. Dickson. The beneficial results of his efforts in the matter of the amended *Workers' Compensation Act* was only one example of what the Association was doing for its members. The meeting had been one of the best held for a long time, and the fact that the convener of the Science Subcommittee had realized the importance of such discussions to the profession and promised to see that there would be more of them would be appreciated by members generally.

VICTORIAN BRANCH NEWS.

THE following items of news, of particular interest to members of the Victorian Branch of the British Medical Association, are published at the request of the Council of the Branch.

"What becomes of my subscription?" This question is commonly heard, and an analysis may be of interest. Each member costs the Branch the following amounts:

	£	s.	d.
The British Medical Journal	1	5	6
THE MEDICAL JOURNAL OF AUSTRALIA	1	0	0
Federal Council—capitation fee	0	2	0
Rebate to country subdivisions (average over all subscriptions)	0	1	6
Debenture interest	0	5	0
Postages, telephones, stationery, lighting, rates, insurances, repairs, sundries	0	5	0
Library, including salaries of library staff	0	4	6
Caretaker	0	1	6
	£3	5	0

The above statement does not include any part of the salaries and travelling expenses of the secretarial and clerical staffs, and if the Branch was not substantially assisted by monetary grants from the British Medical Insurance Company there would be difficulty in carrying on its present activities.

Sickness Incidence and Friendly Societies.

Recent discussions with representatives of the Friendly Societies' Association have shown that they are gravely concerned at the amounts disbursed as "sick pay" in recent years.

The increase in claims and payments is out of all proportion to the growth in membership and its effect on the financial position of the societies, as shown by the most recent quinquennial valuation of the government statist, is serious. In the case of one society, for the five-year period 1922-1926 sick payments averaged £43,700 *per annum*, for 1927-1931 they rose to £50,817 *per annum*, and for the past five years, 1932-1936 have further increased to £64,593 *per annum*—a rise in the last ten years of nearly 48%. The greatest increase in this society has occurred in what might be called the "permanent rate" of 10s. per week, which shows an aggregate rise of 73% in the past ten years. Sick pay for all friendly societies in Victoria has risen from £175,455 *per annum* in 1927 to £249,941 in 1935, an increase of over 42%; yet the increase in membership for the same period is not quite 3%.

Whatever the causes of this drain on the societies' funds, and probably among them can be numbered the financial depression, the difficulty of securing employment *et cetera*, the friendly societies particularly wish the attention of lodge medical officers to be called to the following points and seek their cooperation and assistance in the prevention of malingering and imposition:

That to be entitled to sick pay, a member must be unable to follow any occupation and not merely his usual occupation.

That sick pay is not payable solely on account of old age. It is not a pension. For a pension a very much higher rate of contribution would necessarily have to be charged.

That certificates should not be issued without the medical officer having personally seen the patient at the time of issue and satisfied himself that the member is definitely unable to work.

That certificates should not be ante-dated or post-dated.

That every care should be exercised in certifying only perfectly sound persons as candidates for membership.

Fees for Cremation Certificates.

The Branch Council, in acceding to a request of the Australian Pensioners' League, recommends to members that the fee for the second cremation certificate for old age and invalid pensioners should not exceed £1 1s.

A Tragic Mistake.

An inquest was held by the city coroner on March 9, 1937, on the body of a man of twenty-seven years, who died in a private hospital on February 20. Evidence showed that a rectal injection of paraldehyde in normal saline solution had been ordered as a preliminary to operation, and on February 16 a nurse administered rectally a solution of paraldehyde dispensed by a chemist and delivered to the hospital.

During the administration the patient complained of some pain and rejected portion of the solution, but as he appeared to be quite well, tonsillectomy was performed shortly afterwards. Twenty-four hours later the patient was seriously ill, and by that time it had been discovered that a solution of caustic potash, instead of normal saline, had been added to the paraldehyde. Death ensued four days after the injection, and *post mortem* examination showed extensive necrosis and sloughing of the descending colon.

The coroner held that no blame attached to the doctors concerned or to the hospital, but found that the chemist had been negligent. Although this case was one of those examples of human fallibility, difficult to foresee or guard

against, there is another danger in the use of paraldehyde to which attention was called by Robert Hutchison in the following letter to *The British Medical Journal* of April 12, 1930, page 718:

> Not long ago I had occasion to order a dose of paraldehyde to be given to a young woman under my care in hospital. It was necessary to choose the rectal route, and half an ounce was injected along with three or four times its volume of water. The patient complained of great pain after the injection, and, later, considerable sloughing of the rectal mucous membrane took place. On inquiry it was found that the paraldehyde used had been largely converted into glacial acetic acid. I understand that this takes place according to the equation $CH_3CHO + O = CH_3COOH$, and as no water is formed in the reaction the product is glacial acetic acid, and that once a trace of this has been formed it acts as a catalyst, which may finally lead to the conversion of 75 per cent. of the paraldehyde into acid. If the paraldehyde is kept in an airtight bottle there is no risk of such a conversion, but if the bottle is not airtight oxidation will ultimately take place, with the above results.
>
> I confess I had not known of this danger till the above unfortunate occurrence, and I thought that it might be worth while drawing attention to it.

Additions to Library.

The following books have been added to the library of the Medical Society of Victoria since the publication of the catalogue in May, 1936:

Thomas Anwyl-Davies: "The Treatment of Venereal Disease in General Practice", 1935.

H. Bailey: "Demonstrations of Physical Signs in Clinical Surgery", Fifth Edition, 1935.

Hamilton Bailey: "Diseases of the Testicle", 1936.

Hamilton Bailey and N. M. Matheson: "Recent Advances in Genito-Urinary Surgery", 1936.

Harold Balme: "The Relief of Pain: A Handbook of Modern Analgesia", 1936.

H. Banister: "Psychology and Health", 1935.

D. P. Barr: "Lectures on the Glands of Internal Secretion given under the Auspices of the Melbourne Permanent Post-Graduate Committee, July, 1934", 1935.

Raul Piaggio Blanco and Federice Garcia Capurra: "*La Broncografia en el estudio de las afecciones del torax*", 1935.

William Boyd: "The Pathology of Internal Diseases", Second Edition, 1936.

W. E. Boyd: "Research on the Low Potencies of Homœopathy", Monograph I, 1936.

B. H. Burns and V. H. Ellis: "Recent Advances in Orthopædic Surgery", 1937.

C. Latimer Callander: "Surgical Anatomy", 1934.

C. H. Chalmers: "Bacteria in Relation to the Milk Supply: A Practical Guide for the Commercial Bacteriologist", 1935.

Professor Rosolino Colella (editor): "*Annali della Clinica delle Malattie Nervose e Mentali*", Volume VI, 1934-1935.

H. C. Rutherford Darling: "Surgical Nursing and After-Treatment: A Handbook for Nurses and Others", Fifth Edition, 1935.

"Dental Prescriptions and Formulæ", Dental Board of Victoria, 1936.

Leonard Dobbin and John E. Mackenzie: "Salts and their Reactions: A Class-Book of Practical Chemistry", Sixth Edition, 1936.

Carroll William Dodge: "Medical Mycology: Fungus Diseases of Men and Other Mammals", 1935.

Sir James Elliott: "Scalpel and Sword", 1936.

George B. Eusterman and Donald C. Balfour: "The Stomach and Duodenum", 1936.

H. Bayldon Ewan: "The Principles of Treatment for Diabetic Patients", Third Edition, 1936.

Erik Forsgren: "*Über die Rhythmik der Leber-funktion, des Stoffwechsels und des Schlafes*", 1935.

Ross Golden (editor): "Diagnostic Roentgenology", 1936.

Major Greenwood: "Epidemics and Crowd-Diseases: An Introduction to the Study of Epidemiology", 1935.

H. Ernest Griffiths: "Injury and Incapacity, with Special Reference to Industrial Insurance", 1935.

Alfred Hart: "University Reform and Finance. What Is and What Should Be", 1936.

J. D. Hindley-Smith: "Chronic Streptococcal Toxæmia and Rheumatism", 1935.

George W. Holmes and Howard E. Ruggles: "Roentgen Interpretation: A Manual for Students and Practitioners", Fifth Edition, 1936.

L. Emmett Holt, junior, and Rustin McIntosh: "Holt's Diseases of Infancy and Childhood", Tenth Edition, 1933.

Isabel Emslie Hutton: "The Hygiene of the Change in Women (The Climacteric)", 1936.

Laura Hutton: "The Single Woman and her Emotional Problems", 1935.

Chevalier Jackson and Chevalier L. Jackson: "Diseases of the Air and Food Passages of Foreign-Body Origin", 1936.

L. C. Kellogg: "The Anatomy of Surgical Approaches", 1934.

Peter Kerley: "Recent Advances in Radiology", Second Edition, 1936.

D. Harcourt Kitchin: "Legal Problems in Medical Practice", 1936.

S. A. Levine: "Clinical Heart Disease", 1936.

John Maberly: "Aggressive Medicine", 1935.

Jas. B. Macalpine: "Cystoscopy and Urography", Second Edition, 1936.

A. F. MacCallan: "Trachoma", 1936.

G. F. McCleary: "National Health Insurance", 1932.

Malcolm T. MacEachern: "Hospital Organization and Management", 1935.

W. F. Stewart McKay: "Appendicitis: When and How to Operate", 1936.

John Watts Murray: "Examination of the Patient and Symptomatic Diagnosis", Second Edition, 1936.

"Oxford Medicine", by various authors, edited by H. A. Christian. Volume VII, "Psychiatry for Practitioners", 1936.

O. P. N. Pearn: "Mental Nursing Simplified", Second Edition, 1936.

Grantly Dick Read: "Natural Childbirth", 1933.

Ffrangçon Roberts: "The Principles and Practice of X-Ray Therapy", 1936.

W. Robertson: "An Introduction to Hygiene", Second Edition, 1936.

Jean Rostand: "Adventures before Birth", translated by Joseph Needham, 1936.

John A. Ryle: "The Aims and Methods of Medical Science", 1935.

Erwin Schliephake: "Short Wave Therapy: The Medical Uses of Electrical High Frequencies", English translation by R. King Brown, 1935.

Wilfrid Sheldon: "Diseases of Infancy and Childhood", 1936.

Thomas Stephenson: "Incompatibility in Prescriptions and How to Avoid It", Fourth Edition, 1935.

Alan W. Stewart: "A Manual of Practical Chemistry for Public Health Students", Third Edition, 1935.

Halliday Sutherland: "In My Path", 1936.

Frederick J. Taussig: "Abortion, Spontaneous and Induced: Medical and Social Aspects", 1936.

Sir John Thomson-Walker: "Surgical Diseases and Injuries of the Genito-Urinary Organs", Second Edition, 1936, edited by Kenneth Walker.

T. a'B. Travers: "Concomitant Strabismus: An Essay for the Gifford Edmonds Prize in Ophthalmology", 1936.

Th. H. Van de Velde: "Ideal Birth: How to Get the Finest Children", 1935.

S. Weiss, J. P. Grant and A. J. Quimby: "Diseases of the Liver, Gall-Bladder, Ducts and Pancreas", 1935.

L. E. H. Whitby and C. J. C. Britton: "Disorders of the Blood", 1935.

Wu Lien-Teh and others: "Plague: A Manual for Medical and Public Health Workers", 1936.

"Year Book of Dermatology and Syphilology", 1936, edited by F. Wise and M. B. Sulzberger.
"Year Book of General Medicine", 1936, edited by G. F. Dick.
"Year Book of General Surgery", 1936, edited by E. A. Graham.

NOTICE.

THE Medical Secretary of the New South Wales Branch of the British Medical Association has forwarded for publication the following list of books that have been added to the library of the Branch:

"A Textbook of Pharmacology and Therapeutics", Arthur R. Cushny (Eleventh Edition). Lea and Febiger, Philadelphia, 1936.
"Diseases of the Air and Food Passages of Foreign Body Origin", Chevalier Jackson and Chevalier L. Jackson. W. B. Saunders Company, Philadelphia, 1936.
"Roentgenology", Alban Köhler (Second Edition). Baillière, Tindall and Cox, London, 1935.
"An Introduction to Dermatology", E. H. Molesworth. J. and A. Churchill Ltd., London, 1937.
"An Index of Differential Diagnosis of Main Symptoms", edited by Herbert French (Fifth Edition). John Wright and Sons Ltd., Bristol, 1936.
"The Treatment of Fractures", Lorenz Böhler (Fourth Edition). John Wright and Sons Ltd., Bristol, 1936.
"Applied Physiology", Samson Wright (Sixth Edition). Oxford University Press, London, 1936.
"The Modern Treatment of Burns and Scalds", Philip H. Mitchiner. Baillière, Tindall and Cox, London, 1935.
"Orthopædic Surgery", Walter Mercer (Second Edition). Edward Arnold and Company, London, 1936.
"The Foot", Norman C. Lake. Baillière, Tindall and Cox, London, 1935.
"Vascular Disorders of the Limbs", Sir Thomas Lewis. Macmillan and Co., Ltd., London, 1936.
"Glandular Physiology and Therapy", Council on Pharmacy and Chemistry of the American Medical Association. American Medical Association, Chicago, 1935.
"The Anæmias", Janet M. Vaughan (Second Edition). Oxford University Press, London, 1936.
"Principles of Biochemistry", Albert P. Mathews. Baillière, Tindall and Cox, London, 1936.
"Diseases of Children", edited by Hugh Thursfield and Donald Paterson (Third Edition). Edward Arnold and Company, London, 1934.
"Surgical Diseases and Injuries of the Genito-Urinary Organs", Sir John Thomson-Walker (Second Edition). Cassell and Company, Ltd., London, 1936.
"The Extra Pharmacopœia", Volume II, Twentieth Edition, Martindale and Westcott. The Pharmaceutical Press, London, 1935.
"The Extra Pharmacopœia", Volume I, Twenty-First Edition, Martindale. The Pharmaceutical Press, London, 1936.
"Legal Problems in Medical Practice", D. Harcourt Kitchin. Edward Arnold and Company, London, 1936.
"Text Book of Pathology" (Fourth Edition), Robert Muir. Edward Arnold and Company, London, 1936.
"Diseases of the Eye" (Eighth Edition), J. H. Parsons. J. and A. Churchill, 1936.
"Antenatal and Post-Natal Care", F. J. Browne. J. and A. Churchill, 1935.
"Therapeutic Uses of Infra-Red Rays", W. A. Troup. The Actinic Press, 1936.
"Maternal Mortality and Morbidity", J. M. Kerr. E. and S. Livingstone, 1933.
"Textbook of Roentgenology", B. J. M. Harrison. W. Wood and Company, 1936.
"Theory and Practice of Psychiatry", W. S. Sadler. C. V. Mosby Company, 1936.
"The Toxæmias of Pregnancy", L. McIlroy. Edward Arnold and Company, 1936.
"Infant Nutrition", W. McK. Marriot (Second Edition). C. V. Mosby Company, 1935.
"Life and Works of Lockwood", E. C. O. Dewesbury. H. K. Lewis and Company, Ltd., 1936.
"Minor Practice of General Medicine", L. V. Snowman. John Bale and Sons, 1936.
"Individual Psychology: Theory and Practice", Bevan-Brown, Ward and Crookshank. C. W. Daniel Company, Ltd., 1936.
"Management of Delayed Labour", J. B. Banister. Angus and Robertson Ltd., 1936.
"Study of Masturbation and the Psycho-Sexual Life", S. E. Jelliffe (Third Edition). Baillière, Tindall and Cox, 1936.
"Synopsis of Diseases of the Heart and Arteries", G. R. Herrmann. C. V. Mosby Company, 1936.
"The Woman Asks the Doctor", E. Novak. Christophers, 1936.
"An Ancient Medicinal Remedy", C. J. McAlister. John Bale, Sons and Danielsson, Ltd., 1936.
"Treatment of Fractures in General Practice", Volumes I and II, W. H. Ogilvie. John Bale, Sons and Danielsson, Ltd., 1936.
"Handbook of Surgery", E. C. Mekie. E. and S. Livingstone, 1936.
"Symptoms and Signs in Clinical Medicine", E. N. Chamberlain. J. H. Wright and Sons, 1936.

NOMINATIONS AND ELECTIONS.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Godfrey, Margaret Ann Holst, L.R.C.P., L.R.C.S., 1936 (Edinburgh), L.R.F.P.S., 1936 (Glasgow), Sydney Sanitarium and Hospital, Wahroonga.
Gordon, Neil Acheson, M.B., 1933 (Univ. Sydney), Pacific Highway, Belmont.
Lyne, Ronald George, M.B., B.S., 1935 (Univ. Sydney), Portland.
Fitzgerald, Charles Edward, M.B., 1929 (Univ. Sydney), 229, Maroubra Bay Road, Maroubra.

The undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Abbie, Andrew Arthur, M.B., B.S., 1929 (Univ. Sydney), 790, Military Road, Mosman.
Babbage, Neville Francis, M.B., B.S., 1935 (Univ. Sydney), 4, Roseville Avenue, Roseville.
Benson, Henry Gordon, M.B., B.S., 1936 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Burfitt, James Fitzmaurice, M.B., B.S., 1933 (Univ. Sydney), 86, Enmore Road, Newtown.
Charlton, Robert Ewing Scott, M.B., B.S., 1937 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Cooley, Geoffrey Glover, M.B., B.S., 1937 (Univ. Sydney), Sydney Hospital, Sydney.
Finigan, Frank O'Donnell, M.B., 1936 (Univ. Sydney), District Hospital, Marrickville.
Flashman, James Alan Froude, M.B., B.S., 1936 (Univ. Sydney), 297A, Edgecliff Road, Woollahra.
Godfrey, Norman George, M.B., B.S., 1936 (Univ. Sydney), Sydney Hospital, Sydney.
Johnston, George Arthur William, M.B., B.S., 1937 (Univ. Sydney), Sydney Hospital, Sydney.
Leggett, Clarence Arthur Campbell, M.B., B.S., 1937 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Maynard, Stanley Clive, M.B., B.S., 1935 (Univ. Sydney), Molong.
Middleton, Geoffrey Campbell, M.B., B.S., 1937 (Univ. Sydney), Sydney Hospital, Sydney.
Moore, Henry Dendy, M.B., B.S., 1937 (Univ. Sydney), Sydney Hospital, Sydney.

McColm, Nevyl Fleming, M.B., B.S., 1935 (Univ. Sydney), Dorrigo.
McIntyre, Archibald Keverall, M.B., B.S., 1937 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Monahan, Bryan Willis, M.B., B.S., 1935 (Univ. Melbourne), Mugga Way, Redhill, Canberra, Federal Capital Territory.
Opitz, Favio Phillip, M.B., B.S., 1928 (Univ. Melbourne), 255, Miller Street, North Sydney.
Pardey, Geoffrey James, M.B., B.S., 1928 (Univ. Melbourne), Prince of Wales Hospital, Randwick.
Parker, Alexander Hayward, M.B., B.S., 1936 (Univ. Sydney), Bulli.
Pro-Copis, George Sandford, M.B., 1936 (Univ. Sydney), Sydney Hospital, Sydney.
Ross, Frederick William, M.B., B.S., 1937 (Univ. Sydney), Lithgow District Hospital, Lithgow.
Suttor, Rawdon Thornton, M.B., B.S., 1937 (Univ. Sydney), District Hospital, Manly.
Thompson, Ann Fraser, M.B., B.S., 1935 (Univ. Sydney), Women's Hospital, Crown Street, Sydney.
White, Weeks, M.B., B.S., 1937 (Univ. Sydney), Sydney Hospital, Sydney.

Post-Graduate Work.

COURSE IN PRACTICAL BIOLOGICAL CHEMISTRY IN SYDNEY.

THE New South Wales Post-Graduate Committee in Medicine announces that a course in practical biological chemistry will be held in the Physiology Department of the University of Sydney from 2 p.m. to 5 p.m. on every Wednesday during Trinity term and for the first two Wednesdays of Michaelmas term. The course will begin on Wednesday, June 9. The fee for the course will be £6 6s. Application, enclosing cheque, should be made immediately to the Secretary, New South Wales Post-Graduate Committee in Medicine, the University of Sydney, Sydney.

Correspondence.

"ENSOL."

SIR: There is a letter in your issue of February 27, 1937, from Dr. R. F. Matters in which statements are made upon which you and your readers are entitled to further information and my comment thereon.

It appears that in November, 1935, Dr. Matters heard of "Ensol" treatment for cancer and "journeyed some hundreds of miles to make personal investigations". "No satisfactory evidence of 'cures' was produced at the clinic" and his findings were confirmed by certain gentlemen of Chicago, Montreal and New York who made "personal statements" to him. Upon this is based the profound deliverance that it is unfortunate "Ensol" has been used in Australia as its "curative qualities are quite inferior to others which are better known". "Indeed it is doubtful whether this treatment has any place in the treatment of cancer at all."

Early one evening in November, 1935, Dr. Matters called on me and presented credentials from the Government of Australia. As I was about to attend a meeting of my clinical staff I agreed to see him afterwards and did so at the General Hospital. I made him welcome, invited him to visit the laboratory and clinic the next morning to see the nature of the research and the patients under treatment. He stated that he had stopped off between trains on the way from Toronto to Montreal and was leaving the city again at midnight for Montreal whence he expected to sail for England. I then invited him to return when he had more time, possibly on his way home if he passed through Canada. We spent perhaps half an hour together. That was the extent of his contact and investigation. It was after working hours when he came. He did not visit the clinic, could not have seen any of the patients and heard nothing from me about "cures". Neither then nor at any time have claims of "cures" been made.

Dr. Matters refers to corroborative evidence from gentlemen named. None of these has ever visited me or made use of "Ensol" clinically. Dr. James Ewing, of New York, one of those named, is interested in my work and sends me tissue from the Memorial Hospital to assist the research. To what extent Dr. Matters went out of his way and how complete was his investigation of "Ensol" is manifest.

Other "findings" "submitted to the South Australian Government as honorary Commissioner for Cancer Investigation in May, 1936," may have had a better basis.

In contrast with this brief and hurried visit of Dr. Matters I am bound to tell you that the only other Australian who has been here, Dr. Neville Davis, of Sydney, spent over two months in the summer of 1936 working in the laboratory and clinic and visiting the affiliated laboratories of the Biochemical Research Foundation of the Franklin Institute at Philadelphia. Dr. Davis became thoroughly acquainted with the nature and use of "Ensol", the various aspects of the research in progress and made his own estimate of its value. Before he came he had used "Ensol" in a series of his own cases. Since his return he has continued to use it, the "Ensol" being sent from the laboratory here. Dr. Davis has written: "No one can work with this new therapeutic agent—ensol—without a recognition of its extreme value in favorably influencing the course of advanced cancer patients."

Anyone in Australia or New Zealand who really wants to learn the value of "Ensol" need not accept any opinion, but may see for himself without going very far. Also, upon application, any physician can secure a copy of the bulletin recently published by the Foundation and inform himself. "Ensol" will speak for itself if given an opportunity. "Truth is the daughter of Time, not of Authority." (Bacon.)

Thanks for your courtesy in publishing this letter. There will be no controversy on my part. I am only concerned to be sure that my laboratory work and clinical results are correct.

Yours, etc.,
H. C. CONNELL,
B.A., M.D., C.M., L.M.C.C.

Kingston,
Ontario,
March 27, 1937.

EPIDEMIC PLEURODYNIA.

SIR: Dr. Doig's article in the April 17 issue of this journal was of particular interest to me. Like him, I was at loss to put a name to the condition at first, relying on his "ever-present refuge", influenza, and later recognizing its epidemicity. Dr. Doig gives a very full and clear account of the symptomatology of the disease to which there is little to add. Of the cases seen by me up to the present date, about thirty would be regarded as typical of the condition described. The first case seen occurred towards the end of December, and in the next few weeks quite a number occurred. It would be interesting to know where the outbreak first started, especially with regard to other parts of Victoria, as our own two towns may be looked upon as situated in a more or less contiguous part of the State. I fully agree with Dr. Doig's description of the symptoms, except that there was a definite tendency, particularly in the adults, for the involvement to be left-sided. This led to a few patients coming in with long faces and the firm conviction that they had "heart disease"

or had had a "heart attack". The pain over the precordium and left side, of fairly rapid onset, embarrassed breathing and a "smothering feeling" in the chest made a careful history-taking necessary before the patients' minds could be put at rest. Others showed typical diaphragmatic involvement with respiratory embarrassment, pain in the left shoulder and epigastric tenderness. A further point of interest was that a few, with quite marked symptoms, were afebrile throughout, and these cleared up quickly. With him, I agree that the prognosis was good, complete recovery occurring without complications or prostration. There has been here, about the same time, a mild epidemic of influenza with gastric symptoms, the latter being quite severe in one or two cases, but these all seemed quite clear and distinct from the epidemic in question. The best response was from the liberal use of plastines and "A.P.C." with rest in bed. The infecting organism would probably be similar to the streptococcus which causes epidemic hiccup. In 1923 Rosenow published his experiments with a number of cases of epidemic hiccup. He was able to isolate a streptococcus which would reproduce the disease in rabbits, dogs and monkeys. I can do no better than quote the last paragraph of his "conclusions".

> The question of the origin and absolute specificity of this streptococcus remains to be determined. The facts that it belongs to the pneumococcus-streptococcus group of organisms normally present in the upper respiratory tract of man, that contagion is usually not demonstrable in this disease, and that the power to produce this condition disappears as new and distinctive invasive powers become manifest from successive animal passage and artificial cultivation, suggest that the peculiar localizing power of the hiccough strain is an acquired property, a phase perhaps in the life cycle of the pneumococcus-streptococcus group of organisms.

Yours, etc.,

GEOFFREY PERN.

7, Raglan Street,
Ballarat,
Victoria,
April 26, 1937.

PHYSICAL EDUCATION.

SIR: Dr. Hembrow's broad review of physical education which appeared in your issue of May 8 is most interesting to those of us who are interested in the playground movement.

The American playground movement, providing as it does physical education for all from infancy to old age, and in all weathers, has long provided the requisite supervised system of physical and moral training. Character is developed far more readily in team play than by individual work.

I venture to ask those who sympathize with Dr. Hembrow's essay and who wish to give practical effect to his recommendation, to obtain, read and digest the work of the late Dr. Gulick, entitled "A Philosophy of Play". In that work the full significance and value of cooperative play under educated supervision are clearly set out.

Those of us who have examined the colossal American system and come into contact with the idealists who are associated with it, need no urge to action. But the most difficult practical task with which I am acquainted is on return to Australia to explain such a system in such fashion as to initiate action. Even with graphs and lantern slides *et cetera* the message is received with polite interest and there to a large extent the matter ends.

It is true that those of us who are so interested have secured more than 180 small playgrounds in Melbourne, but they provide only for children and are only under trained direction in some half-dozen instances, thanks largely to the far sight of the South Melbourne City Council. But there is so far not a single large ground conducted on the American plan and providing for people of all ages and at all times. Such a ground is being contemplated at the present time.

The campaign has been conducted for twenty-four years without cessation. If the medical profession took up the problem seriously, much more could and would be done. It is essentially a medical problem in the broadest sense.

Yours, etc.,

JAMES W. BARRETT,
President, Playgrounds Association of Victoria.

103-105, Collins Street,
Melbourne,
May 17, 1937.

Congress Notes.

THE Executive Committee of the fifth session of the Australasian Medical Congress (British Medical Association), to be held at Adelaide in August, 1937, has forwarded further information regarding concessions allowed to members, their wives and families, travelling to Adelaide by air for the congress.

Australian National Airways Proprietary Limited have amended the concessions which they are prepared to grant, as they had been under the impression that concession rates were to apply to members travelling from Adelaide to another State. They are now willing to allow a concession of 10% to individual members, their wives and families travelling to Adelaide from any State excluding Queensland.

The Executive Committee has approached Air Lines of Australia with a view to obtaining a similar concession for members travelling by air from Queensland.

Obituary.

MABEL BAILLIE.

WE regret to announce the death of Dr. Mabel Baillie, which occurred on May 16, 1937, at Carrum, Victoria.

PERCY NEWALL WHITEHEAD.

WE regret to announce the death of Dr. Percy Newall Whitehead, which occurred on May 17, 1937, at Sherbrooke, Victoria.

PORTRAIT OF PROFESSOR W. A. OSBORNE.

IT is the desire of the many friends and old students of Professor Osborne that his portrait should be painted and presented to him, and a commission has been placed with Mr. McInnes. A subscription list has been opened, the subscriptions being fixed at half a guinea. Any friends and old students wishing to be associated with this recognition of Professor Osborne's long services to the University of Melbourne are invited to address cheques to one of the undersigned, Medical Society Hall, 426, Albert Street, East Melbourne, C.2.

B. T. ZWAR.
H. B. DEVINE.
ALAN NEWTON.
VICTOR HURLEY.
SIDNEY SEWELL.

Additional subscriptions have been received from the following: H. F. Maudsley, A. W. D. Robertson, F. G. Middleton, Henry Searby, J. S. Ormond, J. H. Kelly, W. E. Wilson, K. Stuart Cross, G. G. Glassford, B. Hiller, S. O. Cowen.

The total amount received is now £111 6s.

Books Received.

THE CONTROL OF BOVINE TUBERCULOSIS IN MAN, by N. Raw, C.M.G., M.D., M.R.C.P.; 1937. London: Bailliere, Tindall and Cox. Crown 8vo, pp. 135, with illustrations. Price: 6s. net.

EMANOTHERAPY, by F. H. Humphris, M.D., F.R.C.P., D.M.R.E., and L. Williams, M.D.; 1937. London: Bailliere, Tindall and Cox. Demy 8vo, pp. 198, with illustrations. Price: 7s. 6d. net.

DIATHERMY, INCLUDING DIATHERMOTHERAPY AND OTHER FORMS OF MEDICAL AND SURGICAL ELECTROTHERMIC TREATMENT, by E. P. Cumberbatch, M.A., B.M., D.M.R.E., F.R.C.P., with nine collaborators; Third Edition; 1937. London: William Heinemann (Medical Books) Limited. Demy 8vo, pp. 592, with illustrations. Price: 21s. net.

HANDBOOK OF PHYSIOLOGY AND BIOCHEMISTRY, by W. D. Halliburton, M.D., LL.D., F.R.C.P., F.R.S., and R. J. S. McDowall, M.D., D.Sc., F.R.C.P.; Thirty-Fifth Edition; 1937. London: John Murray. Medium 8vo, pp. 935, with numerous illustrations in the text, many of which are coloured, and four coloured plates. Price: 37s. 6d. net.

PRIVY COUNCIL MEDICAL RESEARCH COUNCIL. SPECIAL REPORT SERIES, NUMBER 221. REPORTS OF THE HEARING COMMITTEE. V: HEARING AND SPEECH IN DEAF CHILDREN, by P. M. T. Kerridge; 1937. London: His Majesty's Stationery Office. Medium 8vo, pp. 149. Price: 2s. net.

LEAGUE OF NATIONS PUBLICATIONS. QUARTERLY BULLETIN OF THE HEALTH ORGANIZATION, VOLUME V, NUMBER 2: THE FLY PROBLEM IN RURAL HYGIENE; 1936. Geneva, League of Nations Publications Department; Sydney: H. A. Goddard Limited. Medium 8vo, pp. 179.

LEAGUE OF NATIONS PUBLICATIONS. QUARTERLY BULLETIN OF THE HEALTH ORGANIZATION, SPECIAL NUMBER: REPORT OF THE PERMANENT COMMISSION ON BIOLOGICAL STANDARDIZATION; 1936. Geneva: League of Nations Publications Department; Sydney; H. A. Goddard Limited. Medium 8vo, pp. 174.

THE MORPHINE HABIT AND ITS PAINLESS TREATMENT, by G. L. Scott, M.R.C.S., B.A. (Oxon); Second Edition; 1937. London: H. K. Lewis and Company Limited. Crown 8vo, pp. 114, with illustrations. Price: 5s. net.

Diary for the Month.

JUNE 1.—New South Wales Branch, B.M.A.: Organization and Science Committee.
JUNE 2.—Western Australian Branch, B.M.A.: Council.
JUNE 2.—Victorian Branch, B.M.A.: Branch.
JUNE 3.—New South Wales Branch, B.M.A.: Clinical Meeting.
JUNE 3.—South Australian Branch, B.M.A.: Council.
JUNE 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JUNE 11.—Queensland Branch, B.M.A.: Council.
JUNE 15.—New South Wales Branch, B.M.A.: Ethics Committee.
JUNE 16.—Western Australian Branch, B.M.A.: Branch.
JUNE 17.—New South Wales Branch, B.M.A.: Clinical meeting.
JUNE 22.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JUNE 23.—Victorian Branch, B.M.A.: Council.
JUNE 24.—New South Wales Branch, B.M.A.: Branch.
JUNE 24.—South Australian Branch, B.M.A.: Branch.
JUNE 25.—Queensland Branch, B.M.A.: Council.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xviii to xxi.

BRITISH ASSOCIATION OF RADIOLOGISTS, LONDON, ENGLAND: Fellowship Examinations.

FREMANTLE HOSPITAL, FREMANTLE, WESTERN AUSTRALIA: Medical Superintendent.

INSPECTOR-GENERAL OF MENTAL HOSPITALS, SYDNEY, NEW SOUTH WALES: Honorary Psychiatrists.

LEWISHAM HOSPITAL, SYDNEY, NEW SOUTH WALES: Resident Medical Officers.

ROYAL ALEXANDRA HOSPITAL FOR CHILDREN, SYDNEY, NEW SOUTH WALES: Honorary Officers.

YALLOURN MEDICAL AND HOSPITAL SOCIETY, YALLOURN, VICTORIA: Medical Officer.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY Hospital are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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